Periodontics Syllabus

U. S. NAVY DENTAL CORPS

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Periodontics Syllabus

Prepared by
NAVAL GRADUATE DENTAL SCHOOL
National Naval Medical Center
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Foreword

The Navy Dental Corps has traditionally achieved remarkable progress through the dedicated efforts of its officers. This edition of the *Periodontics Syllabus* has been developed to provide timely information that will aid every dental officer in fulfilling his individual responsibility for sustaining professional competence in all specialized areas of dental practice.

Because of the constant changes in dentistry and allied fields today, dental officers must continually revise their knowledge and skills. This publication is intended to assist the members of the Navy Dental Corps in meeting this professional challenge.

D. L. CUSTIS
Vice Admiral, MC, USN
Chief, Bureau of Medicine and Surgery

Foreword

In these days of rapidly advancing techniques and philosophies of health care, it is imperative that all officers of the Navy Dental Corps continue to update their professional knowledge. This is particularly true in the fast changing field of periodontics.

The *Periodontics Syllabus* will assist the general practitioner in maintaining and improving the high level of performance already obtained. It should also serve as encouragement to every dental officer to recognize and treat periodontal disease in its incipient stages.

The Navy Dental Corps intends to maintain leadership in all phases of dentistry. This publication is one approach aimed at achieving this purpose and furthering our primary mission: To promote the dental health of all Navy and Marine Corps personnel.

R. W. ELLIOTT, JR.
Rear Admiral, DC, USN
Assistant Chief for Dentistry
and Chief, Dental Division,
Bureau of Medicine and Surgery

Preface

The *Periodontics Syllabus* has assisted Navy dental officers in the practice of periodontics for more than 20 years. It is directed toward the individual general practitioner, since it is he who must assume the responsibility for treating the vast majority of patients with periodontal disease. The general dentist today must have the knowledge, the skills, and the motivation to manage most of the periodontal problems that confront him. His responsibility for providing total patient care is in no way diminished by the fact that the Navy Dental Corps includes limited numbers of specialists. Only when the general practitioner and the periodontist combine their efforts and experience in preventing and treating periodontal disease will patients receive dental care of the quality that dentistry professes to provide.

The *Syllabus* is not intended as a textbook but as a manual to complement various inservice training programs. It is therefore uniquely geared to the practice of periodontics in the Navy. The hope is that the general practitioner will gain a better picture of the field, including an understanding of certain procedures that would normally be undertaken only by a periodontist.

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Acknowledgments

This edition of the *Periodontics Syllabus* was authored by Gerald M. Bowers, Captain, DC, USN, Joseph J. Lawrence, Captain, DC, USN, and John E. Williams, Jr., Captain, DC, USN, under authority of the Dental Division, Bureau of Medicine and Surgery, Navy Department. The *Syllabus* is a distillation of the knowledge, philosophies, and clinical experience of a number of periodontists of the Navy Dental Corps, some of whom, in addition, are distinguished educators in civilian dental schools.

Grateful acknowledgment is made to all unidentified authors whose research and philosophies are referenced within the text of this book. Acknowledgment is also made to the staff and students, and to the Publications and Learning Resources Divisions, Educational Resources Department of the Naval Graduate Dental School, who have so willingly assisted in the preparation of this syllabus.

The mention of commercial products in the Syllabus *does not constitute an endorsement of these products by the Department of the Navy or the Department of Defense, and any individual product recommendations contained herein are examples only and do not suggest the exclusion of other comparable items or similar products.*

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The periodontium 1

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The following tissues surround and support the tooth and are known collectively as the periodontium:

1. Gingiva
2. Periodontal ligament
3. Cementum
4. Alveolar process

Gingiva

TERMINOLOGY

The gingiva is composed of keratinizing epithelium and connective tissue. The following terminology is used when describing the gingiva (fig. 1-1):

1. *Marginal (free) gingiva:* the portion of the gingiva surrounding the neck of the tooth, not directly attached to the tooth, and forming the soft tissue wall of the gingival sulcus. It extends from the free gingival margin to the free gingival groove.

2. *Free gingival groove:* the shallow line or depression on the surface of the gingiva dividing the free gingiva from the attached gingiva. The free gingival groove often, but not always, corresponds to the location of the bottom of the gingival sulcus.

3. *Attached gingiva:* the portion of the gingiva that extends apically from the area of the free gingival groove to the mucogingival junction. In the absence of inflammation the attached gingiva is clearly defined, except in the hard palate, where there is no clinical demarcation between the attached gingiva and the remaining masticatory mucosa. The attached gingiva is normally covered by keratinized or parakeratinized epithelium that has marked rete pegs extending into the connective tissue. There is no submucosa, and the attached gingiva is tightly bound down to the underlying tooth and bone. It is apparent that this part of the gingiva is designed to withstand the rigors of mastication, tooth brushing, etc.

The width of the attached gingiva varies from less than 1 mm to 9 mm. Teeth that are prominent in the arch, such as the mandibular cuspid and bicuspid, frequently have a narrow zone of attached gingiva. High frenum and high muscle attachments are likewise associated with narrow widths of attached gingiva. It is not uncommon to see patients who maintain healthy gingiva with less than 1 mm of attached gingiva. However, if there is no band of attached

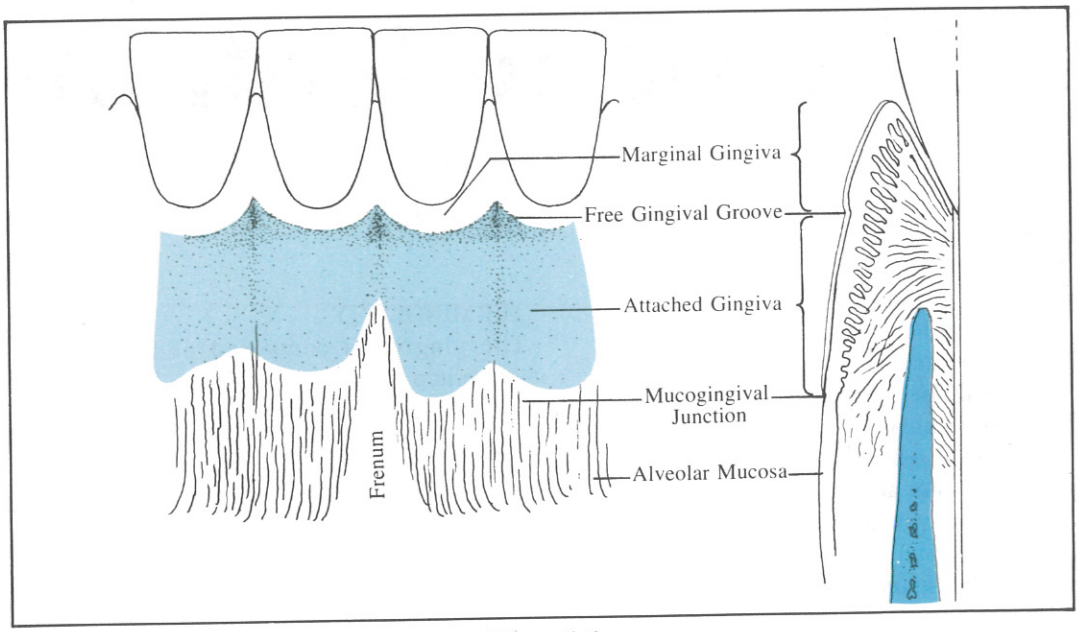


Figure 1-1

gingiva and extension of the lip or cheek results in a pull on the free gingival margin, disease will eventually occur, in the form of plaque infection and/or recession.

An *adequate width of attached gingiva* may be defined, then, as the amount necessary to dissipate the pull of the connective tissue and muscle fibers in the alveolar mucosa.

4. *Keratinized gingiva:* term used to describe the band of keratinized gingiva from the free gingival margin to the mucogingival junction. It is most often used when describing the "detached," attached gingival tissue that remains after periodontal pocket formation. Hence, a patient with periodontal disease may have a broad zone of keratinized gingiva but no attached gingiva (fig. 1-2).

5. *Mucogingival junction:* the scalloped line dividing the attached gingiva from the alveolar mucosa (fig. 1-1).

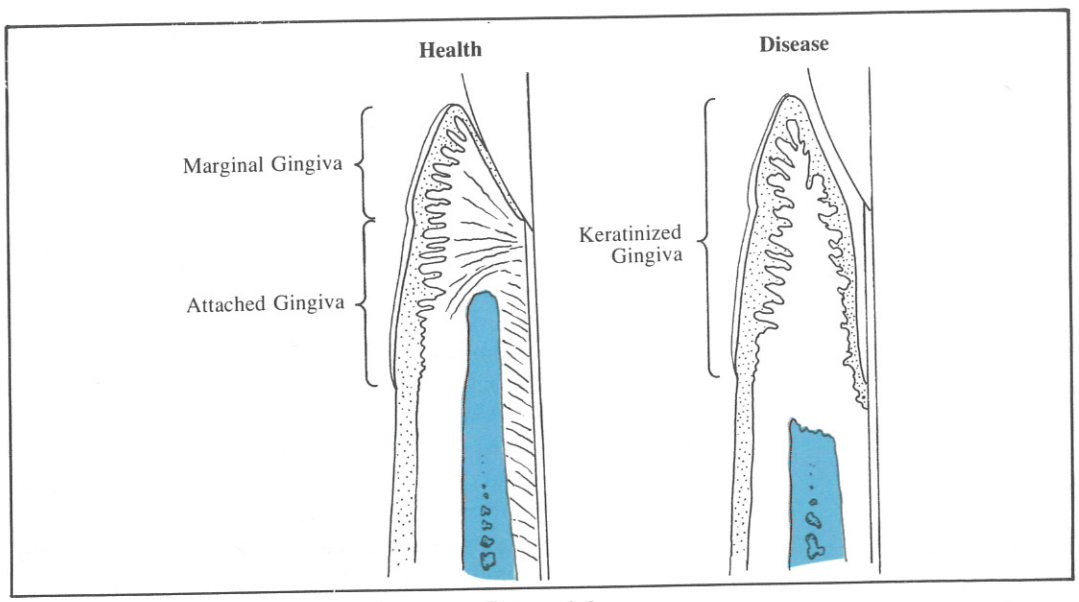


Figure 1-2

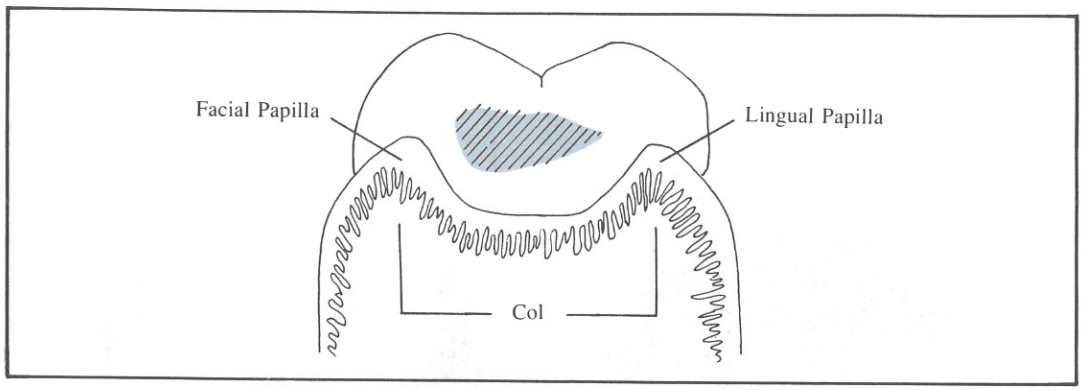


Figure 1-3

6. *Interdental groove:* the vertical groove, parallel to the long axes of adjacent teeth, found in the interdental area of the attached gingiva.

7. *Interdental papilla:* the portion of the gingiva that fills the interproximal space between adjacent teeth. Recently, it has been shown that the interdental papilla is concave, faciolingually. The saddlelike depression has been referred to as the col (fig. 1-3).

8. *Gingival sulcus (crevice):* the space bounded by the tooth and the free gingiva, and having the epithelial attachment as its base.

EPITHELIUM

Gingival epithelium is of the stratified squamous type and, except for that portion lining the gingival sulcus, is parakeratinized or keratinized.

LAMINA PROPRIA

The connective tissue component of the gingiva is termed lamina propria. Like other tissues of the body, it consists of cells (fibroblasts, mesenchymal cells, mast cells, and macrophages), formed elements (collagenous fibers), ground substance (a polysaccharide-protein complex), and a neurovascular network. For the most part, the collagenous connective tissue fibers are oriented into coarse bundles, which are grouped according to location and direction, and are sometimes referred to as the *gingival fiber apparatus*.

GINGIVAL FIBER APPARATUS

1. *Gingival group.* These fibers extend from the cementum in three groups (termed a, b, c), and represent the bulk of the lamina propria facially and lingually (fig. 1-4).

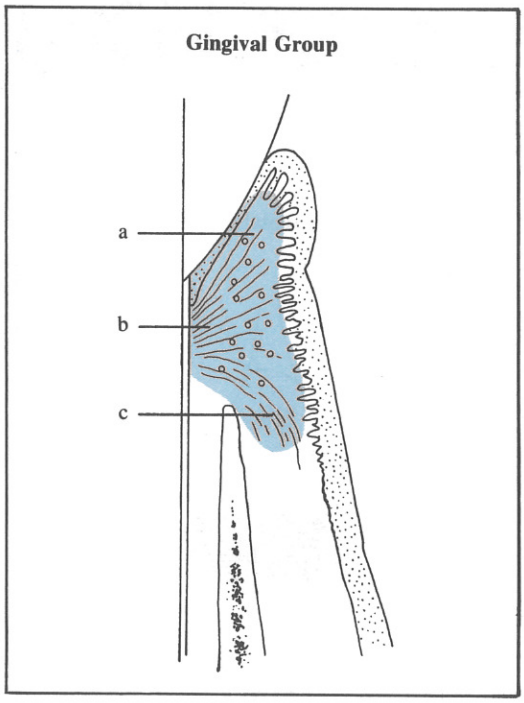


Figure 1-4

2. *Circular group.* The circular fibers encompass the teeth from the margin of the gingiva to the alveolar crest (fig. 1-5).

3. *Transseptal group.* The transseptal fibers extend interdentally from the cementum of one tooth to the cementum of the adjacent tooth. Some authors classify this fiber group with the principal fibers of the periodontium rather than with the gingival fiber apparatus (fig. 1-6).

The basic function of the gingival fiber apparatus is to maintain the free gingiva and epithelial attachment in close approximation to the tooth.

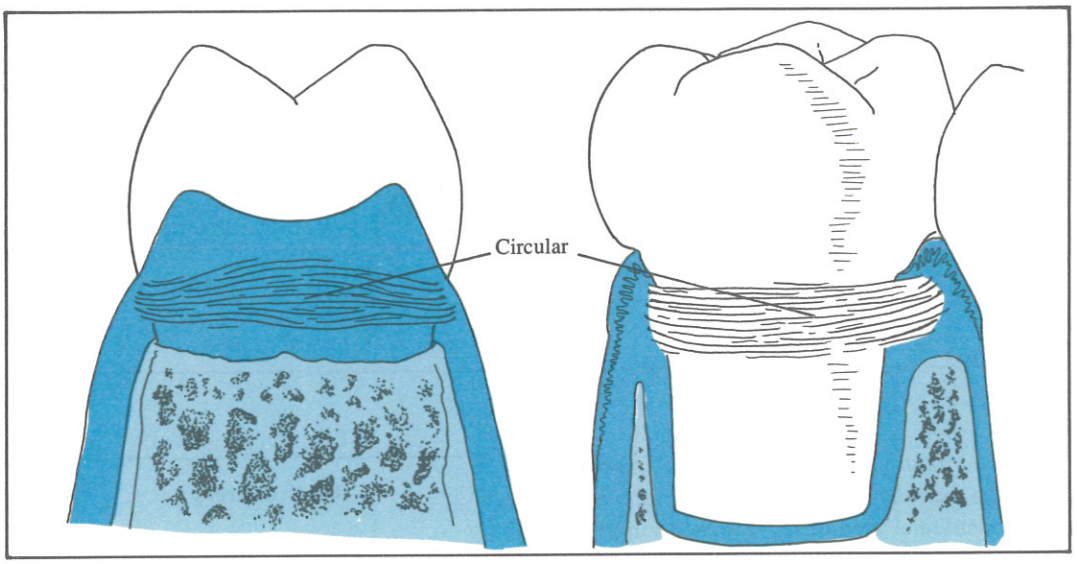


Figure 1-5

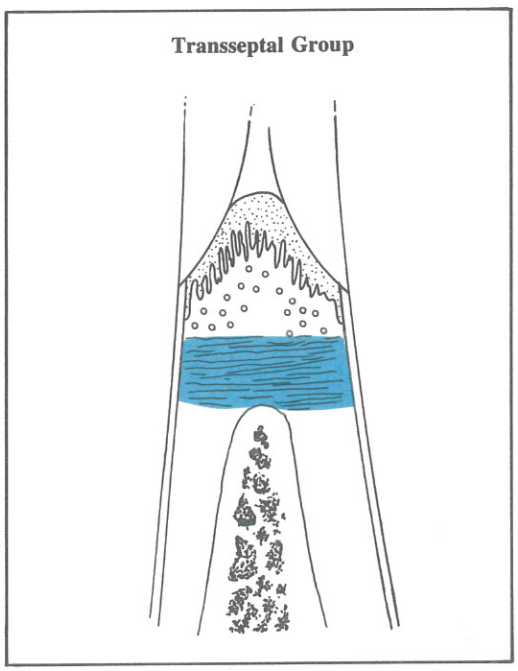


Figure 1-6

ALVEOLAR MUCOSA

The epithelium of the alveolar mucosa is thin, nonkeratinized, and lacks distinct rete pegs. The connective tissue consists of a thin lamina propria and a vascular submucosal layer. The predominant connective tissue fibers are elastic; therefore, unlike the attached gingiva, the alveolar mucosa is loosely bound to the underlying periosteum of the alveolar process. Clinically, the gingiva and alveolar mucosa are separated by the mucogingival junction. On the facial aspects of the maxillary and mandibular arches, the alveolar mucosa extends to the vestibular fornix. On the lingual aspect of the mandibular arch, the arrangement is similar, but there is no demarcation in the maxillary arch. There, the gingiva blends with the palatal mucosa, which is dense and firmly attached to the underlying periosteum. It should be emphasized that the alveolar mucosa is not designed to withstand the forces of mastication and therefore cannot serve as gingival tissue.

CLINICALLY HEALTHY GINGIVA

Several terms used in describing the gingival tissues are important. A concept of what is clinically healthy will enable one to recognize what is abnormal on examination of the gingiva. The following terms are those most commonly used in describing the gingiva:

1. *Color*. The color of normal gingiva is described as coral pink but will vary widely among individuals in shading. The presence of melanin-containing cells (melanocytes) is considered normal for certain ethnic groups such as Negroes, Filipinos, Chinese, etc.

2. *Size*. Any change in size of the gingiva (hyperplasia, inflammation, etc.) is a sign of periodontal disease.

3. *Contour*. This term refers primarily to the festooned appearance of the gingiva.

4. *Consistency*. The gingiva is firm, resilient, and tightly bound to the underlying bone.

5. *Surface texture*. A stippled appearance is normal in the attached gingiva. Loss of stippling may be a sign of periodontal disease. Stippling is caused by projections of the papillary layer of the lamina propria, which elevates the epithelium into rounded prominences that alternate with indentations of the epithelium.

6. *Bleeding on palpation or probing*. Clinically healthy gingiva will not bleed when a periodontal probe is gently inserted into the sulcus or when the marginal gingiva is palpated with the finger.

GINGIVAL SULCUS

The gingival sulcus is lined with a nonkeratinizing stratified squamous epithelium. The bottom of this sulcus is formed by the free surface of the junctional epithelium (epithelial attachment) (fig. 1-7). The sulcus epithelium has been likened to a semipermeable membrane through which injurious bacterial products pass into the gingiva, and tissue fluid (sulcular fluid) seeps out into the sulcus.

JUNCTIONAL EPITHELIUM (EPITHELIAL ATTACHMENT)

The junctional epithelium consists of a collar-like band of nonkeratinizing basal and stratum spinosum-type cells which varies in thickness from 15 to 20 cells coronally, to 1 to 2 cells apically. The cells of the junctional epithelium have relatively wider intercellular spaces and fewer desmosomes when compared with the gingival epithelium. Its location on the tooth depends on the stage of tooth eruption, but it is normally considered to be at or near the cementoenamel junction in the adult. Migration of the junctional epithelium beyond the cementoenamel junction is no longer considered by many to be a physiologic process of aging but rather a pathologic process probably related to repeated plaque infection, tooth position, toothbrush trauma, etc.

The epithelium is attached to the tooth in a manner comparable to that in which the epithelium is attached to the connective tissue of skin or other body surfaces. There is a basal lamina (basement membrane) which consists of two layers, the lamina densa (adjacent to the tooth surface) and the lamina lucida, to which hemidesmosomes (attachment plaques) are attached. A sticky coating (proline and/or hydroxyproline and neutral polysaccharide), which is secreted by the epithelial cells, also binds the junctional epithelium to enamel or cementum.

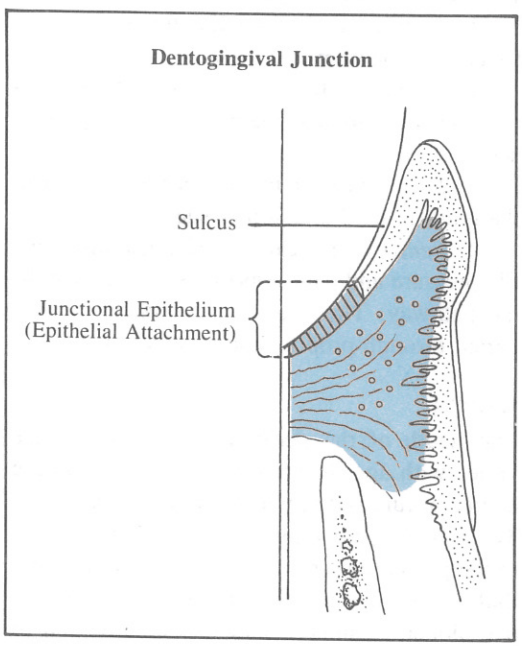


Figure 1-7

DENTOGINGIVAL JUNCTION

The gingival fiber apparatus serves the important function of bracing the gingival and epithelial attachment against the tooth surface. The junctional epithelium and the gingival fibers then act as a functional unit, the dentogingival junction (fig. 1-7).

BLOOD SUPPLY, LYMPHATICS, AND NERVES

Gingival tissue has a rich vascular supply formed by a plexus of arterioles, capillaries, and small veins that extend the entire length of the sulcular epithelium to the outer surface of the gingiva. The blood supply of the gingiva is derived mainly from supraperiosteal branches of the internal maxillary arteries. Vessels from both the alveolar bone and the periodontal ligament merge with the supraperiosteal vessels to form the gingival plexus (fig. 1-8).

The lymphatic drainage of the gingiva begins in the connective tissue papillae and progresses into a network that lies external to the periosteum of the alveolar process. Lymphatics

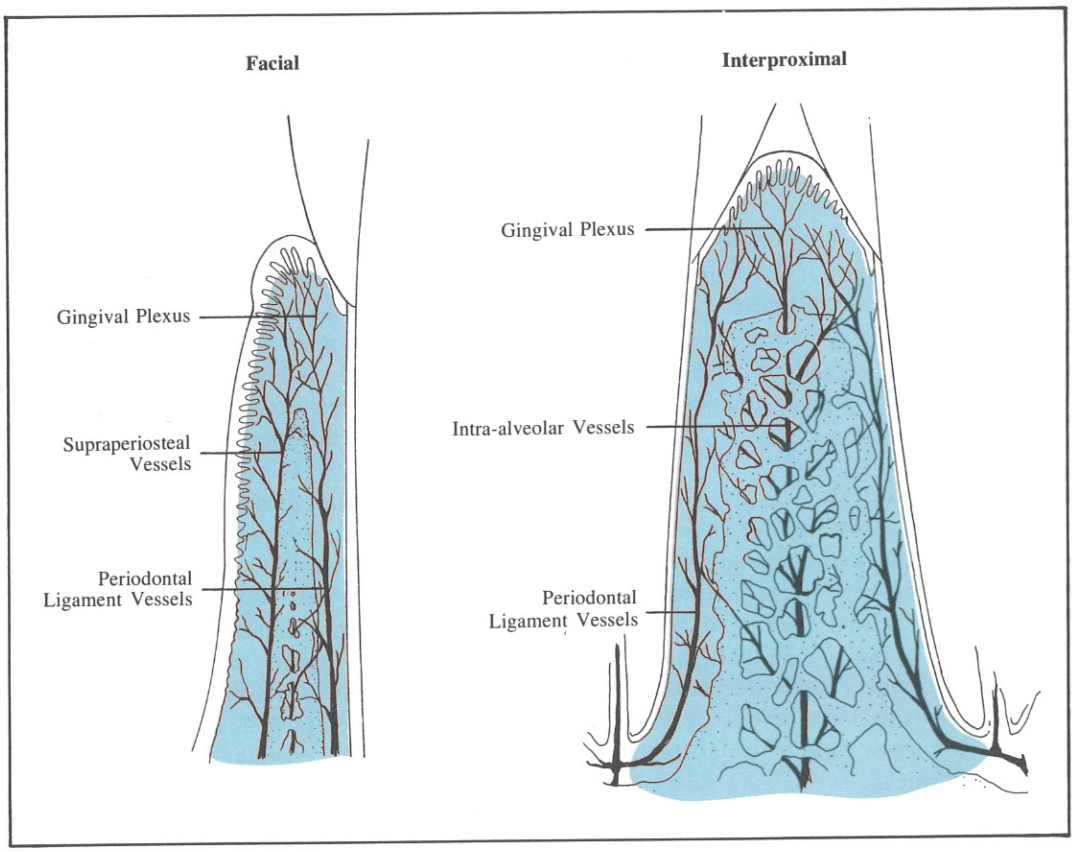


Figure 1-8

drain to regional lymph nodes, particularly the submaxillary group. In addition, lymphatics beneath the epithelium will extend into the periodontal ligament and accompany the blood vessels.

Innervation of the gingiva comes from labial, buccal, and palatal nerves, and from fibers in the periodontal ligament.

Periodontal Ligament

The periodontal ligament comprises the white collagenous connective tissue fibers that surround the root of the tooth and attach to the alveolar process.

There are relatively few elastic fibers in the periodontal ligament. The apparent elasticity is due to the wavy configuration of the principal fibers, which permits slight movement when the tooth is placed under stress.

FUNCTIONS

The functions of the periodontal ligament are:

1. *Formative*. It maintains the biologic activity of cementum and bone.
2. *Nutritive*. It supplies nutrients and removes waste products via blood and lymph vessels.
3. *Supportive*. It maintains the relation of the tooth to hard and soft tissues.
4. *Sensory*. It is capable of transmitting tactile pressure and pain sensations by the trigeminal pathway. The sense of localization is imparted through proprioceptive nerve endings.

WIDTH

The width of the periodontal ligament space varies with age, location on the tooth, and the degree of function to which a tooth is subjected. The mesial side is thinner than the distal, owing to the physiologic mesial drift of teeth. A tooth that is not under function will have a thin periodontal ligament, with loss of direction of the principal fibers. A tooth under normal func-

tion will have a thicker periodontal ligament and a normal configuration of the principal fibers. A tooth in functional occlusion has a periodontal ligament space of approximately 0.25 ± 0.10 mm. A tooth subjected to abnormal stress will have a considerably thicker periodontal ligament space.

BLOOD SUPPLY

The blood supply of the periodontal ligament is derived from three sources:

1. Blood vessels entering the periodontal ligament from the apical area.
2. Interalveolar (interdental) arteries passing into the periodontal ligament from the interdental alveolar process.
3. Anastomosing vessels from the gingiva.

NERVE SUPPLY

The nerves are both myelinated and naked. They vary from knoblike swellings to free endings between fibers.

The nerve bundles follow the course of the blood vessels. Their primary purpose is to transmit proprioceptive sensations via the trigeminal pathways, which give a sense of localization when the tooth is touched.

ATTACHMENT APPARATUS

The attachment apparatus comprises alveolar bone, periodontal ligament, and cementum. The root is attached to bone by numerous bundles of collagenous fibers (principal fibers) that are embedded in cementum and bone (fig. 1-9). These embedded fibers are called Sharpey's fibers. The principal fibers are named according to their location and direction of attachment (alveolar crest, horizontal, oblique, and apical). Interradicular fibers are observed on multirooted teeth. It is still controversial whether principal fibers are continuous from cementum to bone, or whether they are spliced together to form an intermediate plexus.

Cementum

Cementum is the calcified structure that covers the anatomic roots of teeth. It consists of a calcified matrix containing collagenous fibers. The inorganic content is approximately 45 to 50 percent.

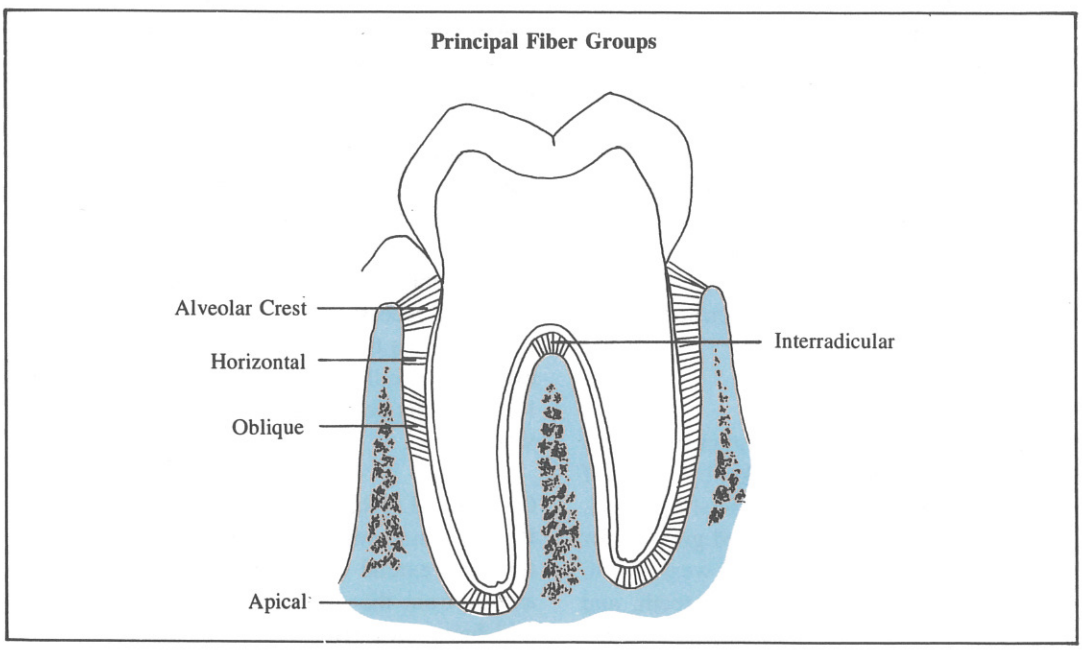


Figure 1-9

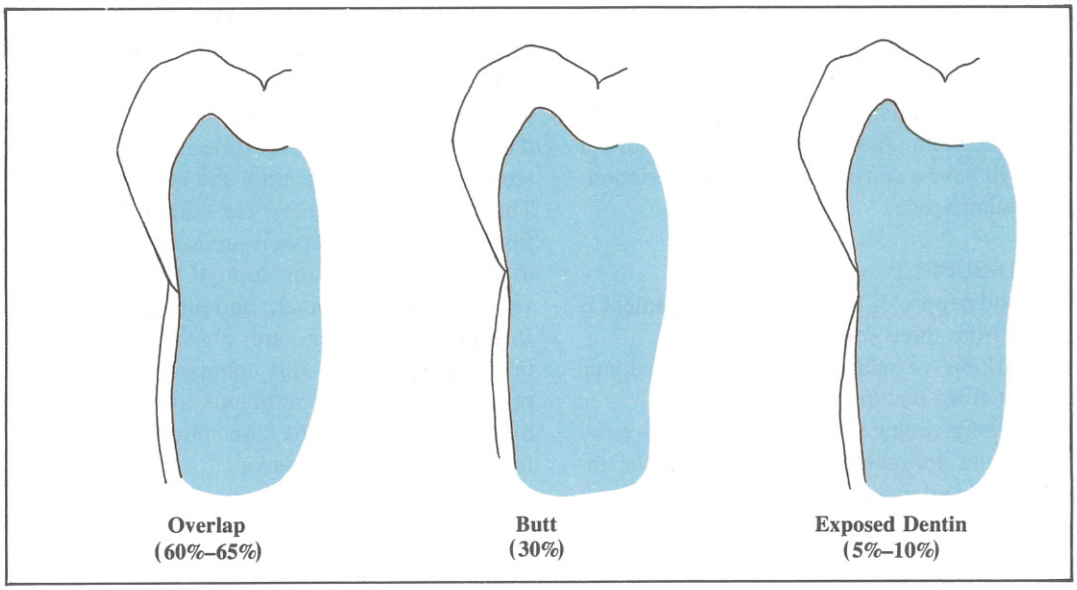


Figure 1-10

CEMENTUM AND CEMENTOID

When first formed, cementum is uncalcified and is known as cementoid. As new layers are formed, the previously formed matrix is calcified and becomes mature cementum. Microscopically, cementum can be divided into two types, cellular and acellular. Functionally, however, there is no difference between the two. The cellular cementum consists of lacunae that contain cells called cementocytes; the cells communicate with one another by means of canaliculi. The distribution of cellular and acellular cementum on the roots of teeth varies. Generally, cementum covering the coronal half of a root is acellular, whereas that covering the apical half is cellular. Cellular cementum is also more prevalent in the bifurcation and trifurcation areas and around the apices of teeth, and is the type of cementum initially formed during wound healing.

FUNCTIONS

The various functions of cementum are (1) to anchor the tooth to the bony socket by means of the principal fibers of the periodontal ligament, (2) to compensate, by its continued growth, for the loss of tooth structure through wear, (3) to facilitate physiologic mesial drift of teeth, and (4) to permit a continual rearrangement of the periodontal ligament.

Cementum is deposited throughout the life of the tooth. The presence of cementoid is considered a barrier to the apical migration of the epithelial attachment and to resorption of the root surface by the surrounding connective tissue.

CEMENTOENAMEL JUNCTION

The relationships of the cementum to the enamel at the cementoenamel junction have clinical significance. There are three types of relationships as demonstrated in figure 1-10. In 60 to 65 percent of the cases the cementum overlaps the enamel, and in 30 percent there is a butt joint. In 5 to 10 percent, however, the enamel and cementum do not meet, and there is exposed dentin. Where this is found, patients may exhibit extreme thermal and tactile sensitivity if recession occurs. Likewise, this defect enhances the accumulation of plaque, with eventual calculus formation. Calculus which forms in this defect defies removal, even when visible.

CERVICAL PROJECTIONS OF ENAMEL

It is not uncommon to observe enamel projections that extend varying distances (grades 1, 2, and 3) from the dentinoenamel junction to the mid-furca area (fig. 1-11). Their role in the spread of disease into the furcation area is un-

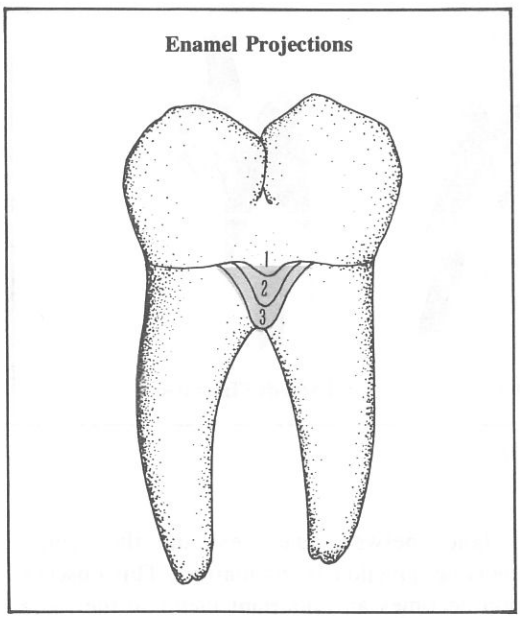


Figure 1-11

known. However, cervical projections of enamel are covered by an epithelial attachment rather than cementum and connective tissue fibers. An epithelial attachment is a potentially weaker attachment than connective tissue and could represent a possible pathway for early involvement of the furcation.

PALATOGINGIVAL GROOVE

Another defect often associated with advanced periodontal destruction is the palatogingival groove (fig. 1-12). This groove is most frequently observed on the maxillary central and lateral incisors, and it often extends from the cingulum to the apex. The palatogingival groove presents a difficult, if not impossible, management problem for the patient and clinician.

Alveolar Process

The alveolar process is that portion of the maxilla and mandible that forms and supports the sockets (alveoli) of the teeth.

DIVISIONS

On the basis of function and adaptation, the alveolar process can be divided into two parts:

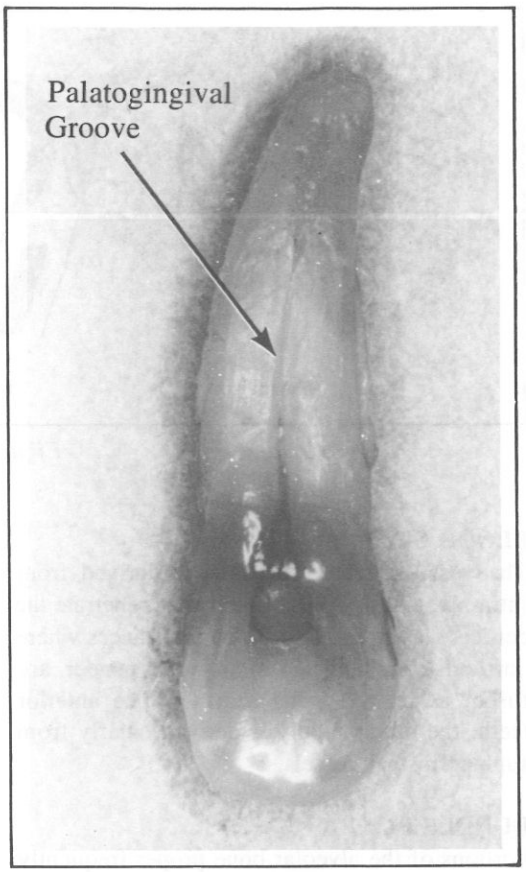


Figure 1-12

1. *Alveolar bone proper:* a thin layer of bone that surrounds the root and gives attachment to the periodontal ligament. This bone is also known as the lamina dura or cribriform plate.

2. *Supporting alveolar bone:* the portion of the alveolar process that surrounds the alveolar bone proper and gives support to the sockets. It consists of:

a. Compact or cortical bone, found on the vestibular and oral aspects of the alveolar process.

b. Cancellous bone (spongy bone), which lies between the alveolar bone proper and the cortical bone. Cancellous bone contains marrow which, in the adult, is mostly of the yellow or inactive type. Foci of red marrow can be found in the maxillary tuberosity and, on occasion, in the maxillary and mandibular molar and premolar areas.

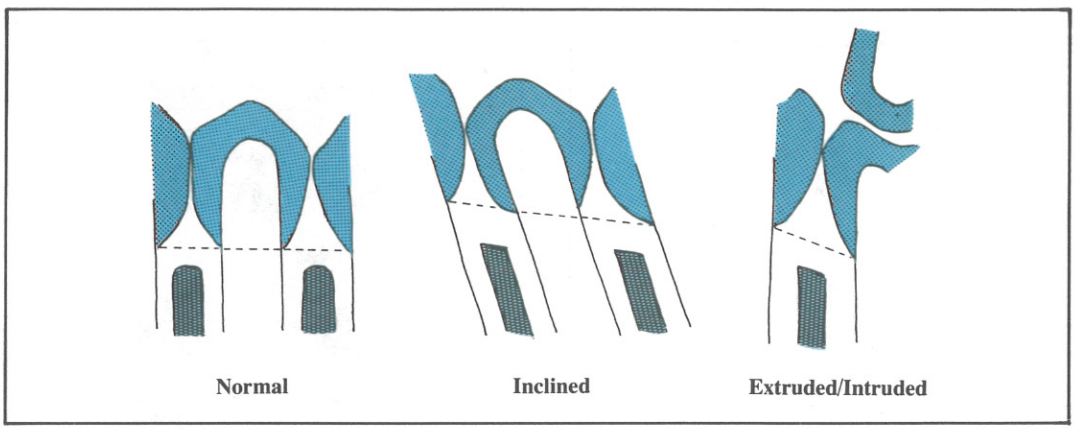


Figure 1-13

BLOOD SUPPLY

The vascular supply of bone is derived from intra-alveolar arteries, vessels that penetrate the cortical plates (fig. 1-8). In circumstances where cortical bone and alveolar bone proper are fused, as on the facial aspect of the anterior teeth, the blood supply is derived chiefly from supraperiosteal vessels.

BUNDLE BONE

Portions of the alveolar bone proper frequently contain "bundles" of calcified collagenous fibers from the periodontal ligament (Sharpey's fibers) and have been termed bundle bone. This is not a special type of bone peculiar to tooth sockets. Bundle bone is present wherever tendons, ligaments, or muscles attach to bone anywhere in the body.

CONTOURS

The contour of the alveolar process conforms to the prominence of the roots and position of the teeth. The height and thickness of the facial and lingual plates are affected by tooth position, root form and size, and occlusal forces. It is not uncommon for teeth that are prominent or in labial versions to bulge through the process, resulting in either an *alveolar dehiscence* or an alveolar *fenestration*.

Teeth that are extruded, intruded, or inclined will have an angular interdental crest (fig. 1-13). Studies have reported that, in health, the alveolar crest will maintain a constant distance from the cementoenamel junction. For example, when a tooth extrudes, bone formation occurs at the alveolar crest, and the distance between the crest and the cementoenamel junction is maintained. This observation becomes an important factor in the radiographic interpretation of infrabony defects.

When the contour of the cementoenamel junction is broad and flat buccolingually (molars and some premolars), the contour of the alveolar process will be broad and flat buccolingually (fig. 1-14). Conversely, the buccolingual contours in the anterior regions are narrow and pointed, owing to the configuration of the cementoenamel junction.

It has been reported that the cervical margin of the alveolar bone is often thickened in response to increased functional demands. This is called *lipping*, or *peripheral buttressing bone formation*.

LABILITY

Alveolar bone is the least stable of the periodontal tissues. It is extremely sensitive to both internal and external stimuli. Its contour and internal structure depend to a great extent on the stresses placed on it.

For instance, in hypofunction, bone is resorbed and there is a decrease in density, with fewer and thinner trabeculae and larger medullary spaces. This is sometimes termed "disuse atrophy." Conversely, in hyperfunction, bone trabeculae are alined in the path of the tensile and compressive stresses, and there is an increase in density. When alveolar support has been weakened by periodontal disease, it is not uncommon to see bone formation occurring either centrally or peripherally in an attempt to buttress the tooth against the occlusal forces

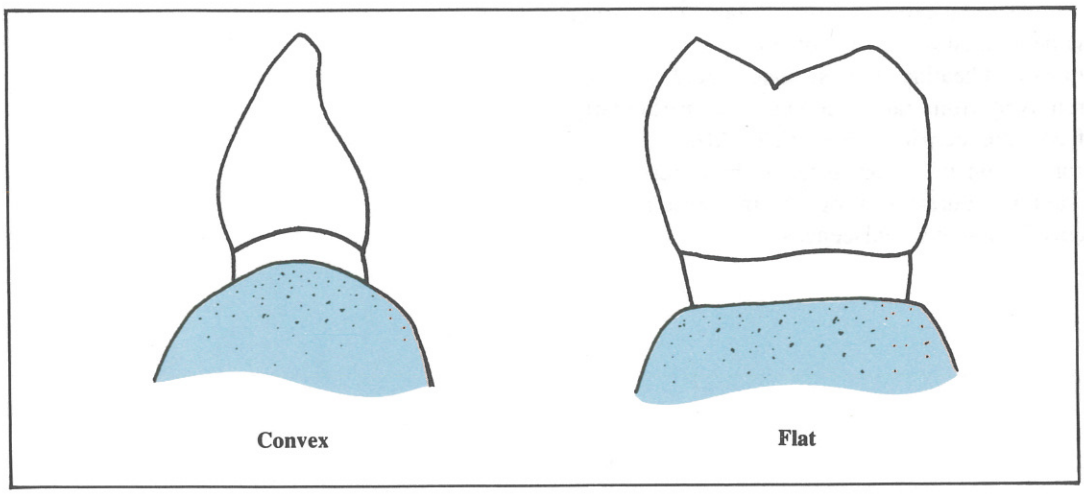


Figure 1-14

(buttressing bone formation). When the attachment apparatus can no longer adapt to occlusal forces, the injury that occurs is termed "trauma from occlusion."

DEHISCENCE AND FENESTRATION

Two defects of the alveolar cortical plate, dehiscence and fenestration, have clinical and therapeutic significance. Dehiscence denotes the cleftlike absence of the alveolar cortical plate, resulting in a denuded root surface. Alveolar fenestration is a circumscribed defect in the cortical plate exposing a facial or lingual root surface (fig. 1-15). Only a very thin layer of combined cortical plate-alveolar bone proper exists over the root surfaces of teeth in labioversion, or over those with large roots. In such cases, there is only minimal, if any, interalveolar blood supply, and blood supply to the bone is derived chiefly from supraperiosteal vessels. The raising of a mucoperiosteal flap and the severing of the supraperiosteal vessels may result in loss of cortical plate, exaggeration of a dehiscence, or a fenestration becoming a dehiscence. When such defects are suspected, every effort should be made to leave connective tissue covering the radicular surface. This is accomplished by utilizing a partial-thickness flap, which preserves the supraperiosteal blood supply.

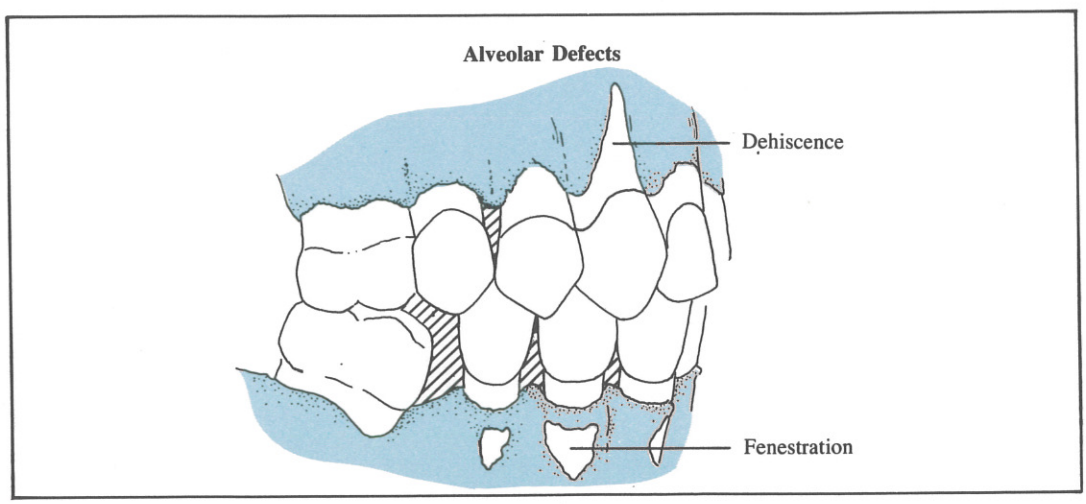


Figure 1-15

A similar problem arises if bone contouring is performed over the root surfaces. According to wound healing studies, bone should rarely be removed from radicular surfaces, particularly from the cervical one-third. After osseous surgery on radicular surfaces, bone resorption continues during healing and may result in extensive osseous dehiscences.

2 Etiology of periodontal disease

Tooth accumulated materials (TAM)
Bacterial plaque
Microorganisms of plaque
Other constituents of plaque
Mechanisms of action
Occurrence of disease
Systemic factors and periodontal disease
Local contributing factors and periodontal disease
Neoplasms

The vast majority of diseases of the periodontium result from bacterial infection. Although other factors may affect the periodontium in one way or another, the dominating causative agents of periodontal disease are colonized microorganisms and their products. Figure 2-1 represents one etiologic classification of periodontal disease. As demonstrated in the drawing, bacterial plaque is directly responsible for the inflammatory diseases of the periodontium, such as gingivitis and periodontitis. There are a number of systemic disorders that adversely affect the periodontium, but no systemic disorder is known to cause chronic destructive periodontitis in the absence of bacterial plaque. Likewise, other local factors act in conjunction with bacterial plaque to produce chronic disease of the periodontium.

Two factors which may initiate periodontal disease in the absence of bacterial plaque are malignancies and primary occlusal traumatism. The role of each factor in the initiation and progression of periodontal disease will be discussed in this chapter.

TOOTH ACCUMULATED MATERIALS (TAM)

In order to discuss bacterial plaque and its relationship to periodontal disease, it is first necessary to define the various materials that accumulate on the tooth surface (Tooth Accumulated Materials, or TAM).

1. *Bacterial plaque:* a mat of densely packed, colonized and colonizing microorganisms, growing on and tenaciously attached to the tooth. Bacterial plaque is not removed with a forceful water spray, but it is readily removed by other mechanical means.

2. *Acquired pellicle:* a thin (0.1 to 0.8 micron), structureless, primarily protein film that forms on erupted teeth and can be removed by

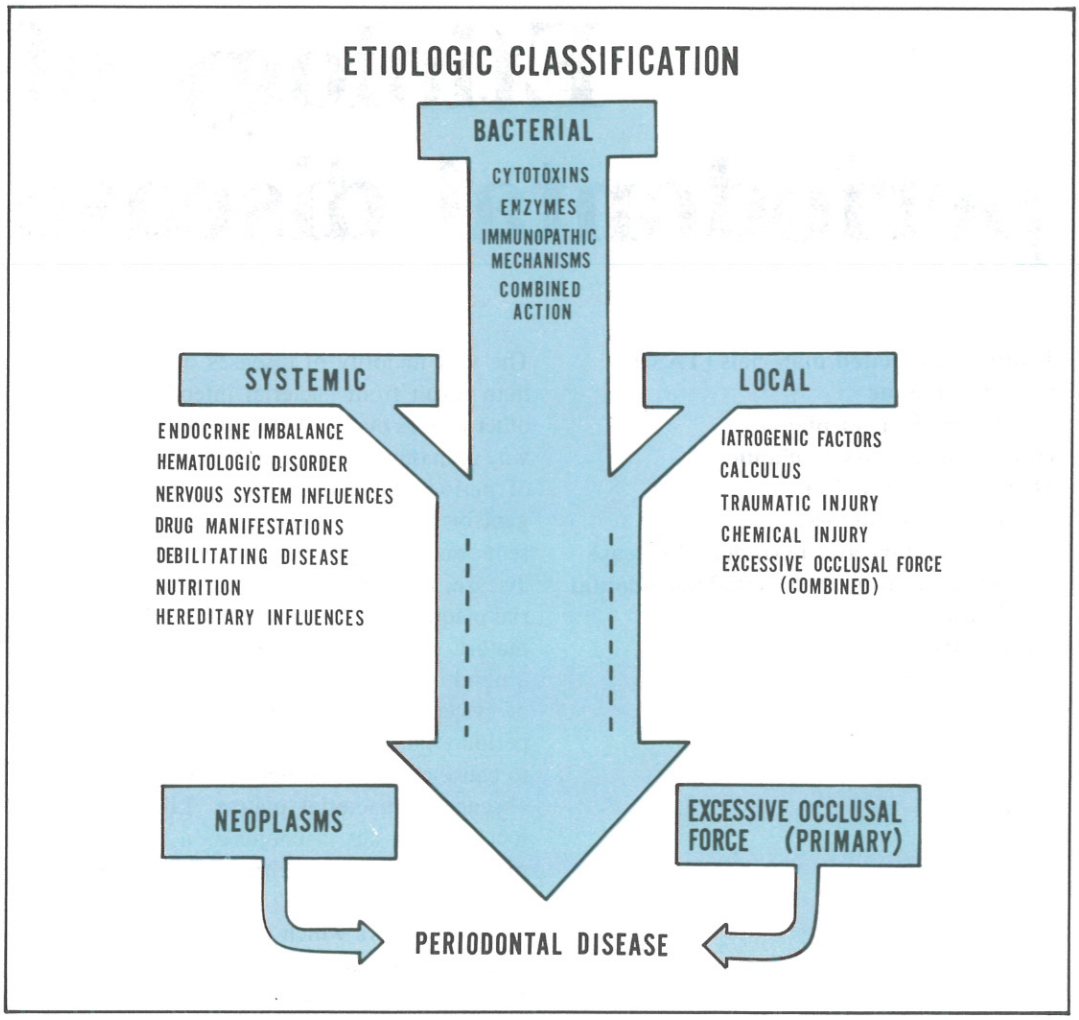


Figure 2-1

abrasives (e.g., pumice). It will quickly re-form after being removed, apparently from constituents of saliva. It can form whether bacteria are present or not. Acquired pellicle will be stained light pink by erythrosin, a red dye commonly used to stain bacterial plaque. Pellicle is not removed by forceful rinsing, and its role in periodontal disease is unknown.

3. *Calculus:* calcified bacterial plaque, usually covered by a soft slime of bacterial plaque.

4. *Food debris:* food retained in the mouth. Debris, unless impacted between the teeth or within periodontal pockets, is usually removed by action of the oral musculature and saliva, or as a result of rinsing.

5. *Materia alba (literally, white matter):* a soft, white mixture of salivary proteins, some bacteria, many desquamated epithelial cells, and occasional disintegrating leukocytes. This mixture adheres loosely to the surfaces of the teeth, to plaque, and to gingiva, and can usually be flushed off with a forceful water spray. The toxic potential of materia alba and its role in the formation of bacterial plaque are not known.

Table 2-1 summarizes some of the differences between plaque, materia alba, and debris.

BACTERIAL PLAQUE

Although many and varied microorganisms normally reside in the oral cavity, their potential for causing damage to the teeth and the periodontium is not realized until they become attached to hard structures, such as teeth, restorations, and orthodontic or prosthetic appliances. Once attached and undisturbed, the microorganisms multiply to form colonies of either the pure or

Table 2-1.—Some Differences between Plaque, Materia Alba, and Debris

Characteristic	Plaque	Materia alba	Debris
Adherence	Close	Loose	None
Effects of rinsing	None	Dislodged by forceful rinsing	Dislodged readily
Structure	Definite	Amorphous	None

mixed type, which are clinically recognizable on a wet tooth as either distinct round colonies or coalesced colonial masses. If the tooth is dried, the masses appear as a dull white film of varying thickness. Unless they are present in massive amounts, colonies may be difficult to see without the most careful scrutiny in good light. Normally white or transparent, they blend with the normal color of the tooth but can be made readily visible by staining with disclosing materials, as will be discussed in chapter 6, "Plaque Control."

MICROORGANISMS OF PLAQUE

The types of microorganisms found in plaque vary among individuals and with the age of the plaque itself.

Young plaque (1 to 2 days) consists primarily of gram-positive and some gram-negative cocci and rods. These organisms normally grow on an amorphous mucopolysaccharide pellicle, less than 1 micron thick, which is attached to the enamel, cementum, or dentin.

From 2 to 4 days, undisturbed plaque changes both in numbers and in types of organisms present. The number of gram-negative cocci and rods increases and fusiform bacilli and filamentous organisms become established.

From 4 to 9 days, this ecologically complex population of microorganisms is further complicated by the presence of increasing numbers of motile bacteria, namely, spirilla and spirochetes.

By the time gingivitis can be diagnosed clinically, the associated plaque will be found to contain all organisms mentioned. Likewise, plaque removed from periodontal pockets will contain a total complex of microorganisms. Some investigators have identified over 300 different species of organisms found within periodontal pockets.

OTHER CONSTITUENTS OF PLAQUE

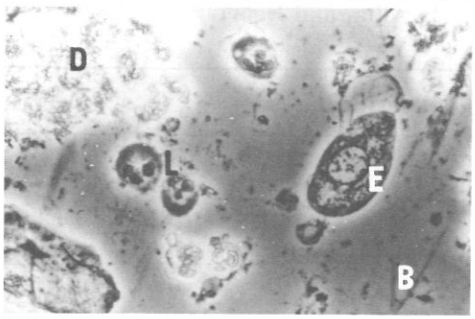
Although colonized organisms are the primary constituents of plaque, additional components can be identified by phase-contrast microscopy. Some of the constituents of plaque are demonstrated in the phase-contrast photomicrograph (figs. 2-2 and 2-3).

1. *Epithelial cells.* These are found in almost all samples of bacterial plaque in varying stages of anatomic integrity. They may range from recently desquamated cells with discrete nuclei and clearly defined cell walls to what may be described as "ghosts" of cells swarming with bacteria.

2. *White blood cells.* Leukocytes may be found in varying stages of vitality in the several stages of inflammation. It is of interest that vital white cells may be found adjacent to clinically healthy gingiva. Living multinucleated leukocytes, their granules vibrating within the cell cytoplasm, present an absorbing sight when viewed with the phase-contrast microscope. Occasionally, pseudopodic movement can be seen as the cells seem to ooze from one position to another. Entrapped microorganisms are often present within the cytoplasm and at times may be observed being engulfed by granulocytes. These vital cells may also be seen in advancing degrees of gingivitis and periodontitis. In areas of obvious exudation and purulence, it is often difficult to find any apparently vital cells among the numerous granulocytes present.

3. *Erythrocytes.* These are readily seen in samples taken from tooth surfaces adjacent to ulcerated gingiva.

4. *Protozoa.* Certain genera of protozoa, notably *Entamoeba* and *Trichomonas,* can often be seen in plaque taken from surfaces adjacent to acute gingivitis and from within periodontal pockets.



D = Debris
E = Epithelial cells
B = Bacteria
L = Leukocytes

Figure 2-2

D = Debris
E = Epithelial cells
B = Bacteria
L = Leukocytes

Figure 2-3

5. *Food particles.* Occasionally, microscopic shreds of food are seen. The most readily recognized are muscle fibers, distinguishable by their striations.

6. *Miscellaneous.* Nonspecific elements, such as crystalline-appearing particles (which may be fine fragments of cementum, beginning calcifications, or unidentified foodstuffs) and what seem to be cell fragments, may also be found in plaque.

MECHANISMS OF ACTION

1. *Invasion.* Whether or not bacteria invade the gingiva is, in the light of present knowledge, a moot point, since bacterial invasion is not necessary to the occurrence of gingival inflammation. It is only necessary that enough bacteria be fixed to the tooth, near the gingiva, for a long enough time to challenge the tissue with their toxic products. No specific organism or group of organisms has been positively or exclusively identified as the cause of periodontal breakdown, but many potentially toxic products of the plaque have been identified.

2. *Cytotoxic agents.*

a. Endotoxins, which are lipopolysaccharide constituents of the cell wall of gram-negative bacteria, can be a direct cause of tissue necrosis. Also, endotoxins from certain oral organisms will stimulate bone resorption in tissue culture.

b. Small metabolites, such as L-aspartic acid, have been recovered in greater quantity from plaque near clinically inflamed gingiva than from plaque near clinically uninflamed gingiva. Application of L-aspartic acid to experimental animals will likewise produce gingival lesions.

3. *Enzymes.*

a. Collagenase—depolymerizes collagen fibers and fibrils, the major formed elements in the gingiva and periodontal ligament. It is of interest that leukocytes, also, are known to produce collagenase and are present in large numbers in the early lesions of gingivitis.

b. Hyaluronidase—hydrolyzes hyaluronic acid, an important tissue-cementing polysaccharide, and can act as a "spreading" factor to increase tissue permeability. This enzyme is produced by both microorganisms and the host.

c. Chondroitinase—hydrolyzes chondroitin sulfate, another tissue-cementing polysaccharide.

d. Proteases—contribute to the breakdown of noncollagenous proteins and increase capillary permeability.

The aforementioned are cytotoxic agents that have been identified, but undoubtedly they do not represent all of the toxic products of plaque microorganisms.

4. *Immunopathologic mechanisms.* Studies have demonstrated that a number of plaque antigens will induce inflammation in animals. Both the humoral and the cell-mediated types of immune response have been observed in patients

with periodontitis. The role of allergic reactions in periodontal disease is not completely understood; however, the potential is apparent.

5. *Combined action.* It is possible that more than one mechanism may be involved in the initiation of periodontal disease. For example, it is conceivable that bacterial enzymes and/or cytotoxic substances exert a direct effect on the sulcular and subsulcular tissue, as well as initiate an indirect immunopathologic response.

The exact mechanism of action of bacterial plaque is unknown; however, there remains little doubt that the vast majority of periodontal disease is bacterial in origin.

OCCURRENCE OF DISEASE

It is not uncommon to find people whose periodontal health is good despite the presence of bacterial plaque, and to find others with marked periodontal disease in the presence of only small amounts of plaque.

Resistance. The general health status of an individual depends on the presence or absence of a cause for disease and on the individual's ability to resist that cause. This concept can be reduced to a formula: R/C = Hs, in which R equals Resistance, C equals Cause, and Hs equals Health status.

It is evident that health will be at a maximum in the absence of cause and at a minimum in the absence of resistance. As R and C vary, so also will Hs. The same formula is applicable to the health of a specific gingival area.

The accumulation of bacterial plaque causes gingivitis. The ability of the plaque to cause disease depends on the number and virulence of the microorganisms and the length of time they remain on the tooth surface. Resistance refers to the ability of a specific gingival tissue to withstand the damaging effects of colonized bacteria contiguous to that tissue. (Although the term "resistance" is easily used and its implications are readily comprehended, the precise mechanisms of resistance, at the molecular level, are unclear.) Health status refers to the presence or absence of inflammation in the tissues considered.

The formula R/C = Hs has meaning only as it increases our understanding of variations between different patients and between different gingival areas in the same patient.

SYSTEMIC FACTORS AND PERIODONTAL DISEASE

Systemic factors related to periodontal disease will be discussed in chapter 3, but it should be mentioned that any condition that might reduce the resistance (R) of the gingiva should be expected to contribute to the initiation of inflammation and to influence the rapidity and severity of the disease process.

LOCAL CONTRIBUTING FACTORS AND PERIODONTAL DISEASE

1. *Iatrogenic factors.* There are a number of procedures, techniques, and materials used in dentistry which indirectly, and on occasion directly, contribute to the initiation and/or progression of periodontal disease:

a. *Operative procedures.* Most injuries to the gingiva that occur during restorative dentistry procedures are of a minor nature and heal readily without loss of form or function of the periodontium. However, some precautions should be observed. For example, if a large mass of papilla is destroyed by careless use of a wedge during matrix stabilization, it is very likely that the papilla will not regenerate to normal contour. Also, retraction cord, impression tubes, diamond stones, and temporary restorations may result in irreversible damage to the periodontium if either of the following conditions exist:

If there is very little attached gingiva at the operative site, it can easily become macerated and detached, with ultimate loss of all attached gingiva. Under such circumstances, loss is almost inevitable if there is a frenum attachment at the preexisting mucogingival junction.

Irreversible damage will result if gingiva is stripped from a tooth and an overextended temporary restoration is placed, or if cementing material is forced between the tooth and the detached tissue and left in place. In either condition, epithelium will attempt to cover the detached tissue and, when the temporary restoration (or cement) is finally removed, a deepened, epithelium-lined pocket will exist. The longer the materials remain interposed between tooth and tissue, the greater the certainty of permanent loss of the gingival attachment.

b. *Restorative materials and restorations.* Except for plastics in which excess free monomer is present, no restorative material used

in dentistry today has been shown to be, in itself, capable of producing inflammation.

Restorations may play a role similar to that of rough calculus if they have overhanging margins or rough surfaces. Since overhanging margins and surface irregularities provide sites for plaque formation and retention, one should be cautious in relating chronic inflammation solely to the direct effect of the restorations. It is more likely that the presence of overhanging margins and rough surfaces makes plaque removal impossible and provides protected areas for microorganisms to eat, multiply, and excrete their toxic products.

c. *Removable partial dentures.* If a prosthesis is so designed as to impinge on the soft tissue or exert torque on the teeth, direct damage to the periodontium can occur. In the presence of bacterial plaque, these insults can result in rapid, severe destruction of periodontal structures.

d. *Fixed partial dentures.* In addition to the necessity for marginal excellence on abutments, there is a further requirement that the design be such that the patient can clean all surfaces of the restoration. This requirement suggests open interproximal embrasures and generally convex surfaces to enhance cleansability of the prosthesis. These principles are especially critical in pontic design. Failure to instruct patients in the methodology of cleansing fixed partial dentures is the first step toward eventual breakdown of the periodontium.

e. *Exodontics.* When extractions are performed so that the attachment apparatus of an adjacent tooth is damaged at or near the dentogingival junction, the damage is frequently irreversible. For example, the soft tissue and bone supporting an adjacent tooth can be destroyed if they are injudiciously used as a fulcrum for a surgical elevator. Poor flap design, as well as poor approximation and fixation of wound edges, can result in tissue contours which are conducive to plaque and food retention. Failure to remove calculus from adjacent teeth at an extraction site will negate an excellent opportunity for pocket elimination and regeneration of the attachment apparatus around the remaining teeth. This oversight indirectly enhances the progression of periodontal disease.

f. *Orthodontics.* Fixed appliances (bands, wires, etc.) present excellent harbors for bacterial growth and can thus contribute significantly to inflammation. Temporary extracoronal splints, whether composed of welded orthodontic bands, wire, or wire and acrylic resin, may also be included in this category.

On final analysis, it is evident that poor dentistry of all types may create sites for the formation of plaque, intensify its production, and prevent its mechanical removal.

2. *Calculus.* Calculus is calcified dental plaque. It should not be considered a direct cause of inflammation except in those rare instances where soft tissue is abraded by being forced against a rough edge of calculus. Even in such instances, it would be difficult to attribute chronic inflammation to the calculus itself, since it is normally covered by a soft slime of bacterial plaque.

It is possible that calculus is important in the progression of disease, serving as a "coral reef" within which microorganisms can multiply and excrete. The rough surface of calculus makes it difficult, if not impossible, for the patient to remove bacterial plaque.

In any case, there is ample evidence that complete removal of calculus is necessary for resolution of periodontal pockets.

3. *Traumatic injuries.* Traumatic injuries to the periodontium can result in the loss of the attachment apparatus and contribute to the initiation and progression of periodontal disease.

a. *Toothbrush abrasion.* This can completely destroy a narrow band of attached gingiva and result in extensive recession. In fact, toothbrush abrasion is one of the two most common factors associated with recession, the other being tooth position. Such abrasion also results in extensive grooving of the root surfaces, which causes cleansing problems for the patient and management problems for the dentist.

b. *Factitious disease.* Occasionally, patients are encountered who persistently gouge or "scratch" their gingiva with their fingernails (factitious disease). This usually results in extensive exposure of the root surface and localized inflammation. This rare entity is a difficult diagnostic problem. Whenever isolated areas of recession are noted and a thorough evaluation fails to identify the etiology of the disease, factitious disease should be considered.

c. *Food impaction.* This is one of the more common local factors which contribute to the initiation and progression of periodontal dis-

ease. Open contacts, uneven marginal ridges, irregular positions of teeth, and nonphysiologic contours of teeth and restorations can result in the impaction of food on the gingiva and into the gingival sulcus. Some investigators believe that food impaction is an important factor in vertical bone loss. It is not clearly understood what produces the initial breakdown in an area of food impaction or food retention. It can be speculated that the forceful wedging of food beneath the gingival tissues can produce inflammation from physical trauma, in addition to tearing of the epithelial attachment. It is just as likely, however, that the initial injury is a result of food degradation and chemical irritation. It is also possible that food impaction and retention merely afford an excellent breeding ground for bacteria that initiate the disease process.

4. *Chemical injury.* Indiscriminate use of aspirin tablets, strong mouthwashes, and various escharotic drugs may result in ulceration of the gingival tissues. Likewise, dentists may inadvertently permit strong bleaches or salts of heavy metals, such as silver nitrate, to come in contact with the tissue. Injuries of this nature are usually transient but may contribute to the destruction of periodontal tissues or the initiation of plaque infection if the insult becomes chronic in nature.

5. *Excessive occlusal force.*

a. *Combined periodontal traumatism.* When excessive occlusal force is directed on a diseased periodontium, the disease process can be exaggerated. This combination of excessive force and existing inflammation is called combined periodontal traumatism. For example, in a patient with generalized moderate periodontitis, it is not uncommon to observe extensive destruction of one or two teeth which are subjected to parafunctional habit patterns of bruxing, clenching, doodling, or fingernail biting. In this instance, the persistent and often excessive occlusal force generated by habit is a codestructive factor working in conjunction with an existing plaque infection to produce an exaggerated loss of the attachment apparatus. The relationship of combined periodontal traumatism and periodontal disease will be discussed in greater detail in chapter 21.

b. *Primary periodontal traumatism.* This condition is characterized clinically by tooth mobility, pain on chewing, tenderness to percussion, and thermal sensitivity in the *absence* of gingival inflammation and pocket formation. Microscopically, the periodontal ligament may demonstrate necrosis, hemorrhage, thrombosis of blood vessels, dissolution of principal fibers; and there is bone loss and widening of the periodontal ligament space. Primary periodontal traumatism is usually a result of excessive force associated with high restorations, removable partial dentures that traumatize abutment teeth, drifting of teeth following extraction, and parafunctional habits. It should be reemphasized that there is *no* pocket formation and the lesion can usually be corrected by eliminating the local etiologic factor and/or adjusting the occlusion.

NEOPLASMS

There are a number of benign and malignant lesions that involve the tissues of the periodontium. It is not within the scope of the *Syllabus* to discuss the various neoplasms of the periodontium, and the reader is referred to a standard text in oral pathology for this information.

3 Systemic contributing factors in periodontal disease

Systemic factors
- Endocrine imbalance
- Hematologic disorders
- Nervous system influences
- Drug manifestations
- Debilitating diseases
- Hereditary influences
- Nutrition
  - Relationship to periodontal disease
  - Nutritional factors
  - Nutritional deficiencies
  - Tissue changes related to nutritional deficiencies
  - Evaluating the nutrition

Systemic Factors

Any systemic condition that markedly alters cellular metabolism may change the response of the periodontal tissues to irritants and alter their healing and regenerative capacity. For example, it is not uncommon to see patients with some systemic disease (diabetes mellitus, leukemia, anemia) demonstrate an exaggerated gingival response to minimal plaque accumulation.

A few systemic factors that have been shown to adversely affect the periodontal tissues are as follows:

1. Endocrine imbalance.
2. Hematologic disorders.
3. Nervous system influences.
4. Drug manifestations.
5. Debilitating diseases.
6. Hereditary influences.
7. Nutrition.

ENDOCRINE IMBALANCE

In an uncontrolled diabetic, the gingiva may be deep red, edematous, slightly enlarged, and prone to abscess formation. Loosening of teeth and extensive loss of supporting structures may occur in a relatively short time. The rate of healing is markedly delayed and resistance to infection is lowered. Periodontal treatment is contraindicated for patients with uncontrolled diabetes, but not for patients whose disease is under control. It is important to consult with the attending physician of all diabetic patients prior to periodontal therapy. All surgical procedures

should be performed as atraumatically as possible, preferably 2 to 3 hours after meals or after the administration of insulin.

Hormonal changes that occur during puberty, menstruation, or pregnancy alter the periodontal tissues to the extent that an irritant such as bacterial plaque, which initially produces a somewhat mild reaction, may elicit an exaggerated inflammatory response. Changes that may occur in the gingiva during puberty or menstruation are hyperemia, pain, swelling, and hemorrhage. Changes during pregnancy may vary from a mild gingivitis to a so-called pregnancy tumor. A pregnancy tumor is a localized, inflammatory, hyperplastic lesion usually arising from the gingiva in the vicinity of an interdental papilla or other area of irritation or infection. Treatment of patients with hormonal imbalance usually consists of the removal of local irritating factors and instruction in plaque control. It is imperative that patients understand the grave consequences if meticulous plaque control is not carried out during periods of hormonal change.

HEMATOLOGIC DISORDERS

Leukemia, the malignant overproduction of leukocytes, is apparently on the increase. The oral lesions of acute leukemia consist of marked gingival hyperplasia, ulcerations of the gingiva, and persistent bleeding. Patients with such oral lesions may seek dental treatment before the disease has been diagnosed. In the presence of acute or subacute leukemia, the treatment of periodontal disease is limited. Treatment should be directed toward relieving pain and maintaining the best possible plaque control. Surgical procedures are contraindicated, as they may result in necrosis, severe hemorrhage, and rapid death.

Other forms of blood dyscrasia in which periodontal treatment is limited are hemophilia, agranulocytosis, thrombocytopenic purpura, and polycythemia. The various anemias may also be severe enough to impair normal cellular metabolism and thereby exert an influence on the progress and treatment of periodontal disease.

NERVOUS SYSTEM INFLUENCES

Autonomic nervous system influences may cause local alterations in blood supply and thereby impair tissue nutrition. Autonomic influences may also affect the periodontium by eliciting changes in salivary flow.

Psychological problems may cause occlusal neuroses, interfere with oral hygiene and diet, or initiate undesirable oral habits. The high incidence of periodontal disease in patients with mental disorders is probably related to the inadequate plaque control generally associated with these patients.

DRUG MANIFESTATIONS

Reactions to certain drugs produce changes in the gingiva:

1. Ingestion of heavy metals, such as bismuth, lead, and mercury, produces a linear pigmentation in the gingiva.
2. Diphenylhydantoin sodium, used in the treatment of epilepsy, is frequently associated with marked gingival hyperplasia (dilantin hyperplasia). Hyperplastic changes appear first in the interdental papillae, and in advanced cases the teeth may be completely covered.
3. Systemic administration of cortisone and other adrenal corticosteroids lowers the patient's ability to cope with infections and impairs healing.

DEBILITATING DISEASES

As previously stated, the response to irritants and the healing and regenerative capacities of periodontal tissues may be changed by systemic conditions that markedly alter cellular metabolism. Consequently, any debilitating disease, as well as normal aging, may adversely affect the periodontal tissues and their responses.

It is evident, therefore, that some systemic factors play a vital role in the progress of periodontal disease and influence its treatment. When systemic disturbances are present, the treatment of periodontal disease is dictated by the condition. In most systemic related disturbances, elimination of local irritants will reduce inflammation, but elimination or control of the systemic disease is necessary for restoration to complete health.

HEREDITARY INFLUENCES

Heredity may be a factor in periodontal disease. At least, studies in experimental animals indicate the possible relationship. There are a number of genetic disorders which affect the

oral mucous membrane, and some studies suggest the inheritance of a predisposition or tendency toward periodontal disease. As yet, however, there is no conclusive evidence that heredity plays a role in the initiation and progression of periodontal diseases in man.

NUTRITION

Relationship to Periodontal Disease

To a large extent, healthy periodontal tissues are dependent on utilization and absorption of the nutrients obtained in a balanced diet. No cause-and-effect relationship between specific nutrients and periodontal disease has been adequately shown in humans. The periodontist, however, is vitally concerned with epithelial keratinization, vascular integrity, osteogenesis, wound healing, and tissue repair—all of which are directly related to nutrition at the cellular level.

Nutritional Factors

Nutrition consists of taking in and assimilating carbohydrates, fats, proteins, vitamins, and minerals, from which tissue is built up and energy liberated. It involves the processes of digestion, absorption, assimilation, and excretion. When nutrients necessary for normal tissue functions are lacking, a nutritional deficiency will occur after sufficient time has elapsed to deplete the body's nutrient reserve. When this stage has been reached, biochemical differences, functional changes, and anatomic lesions will occur.

Nutritional Deficiencies

Nutritional deficiencies may be (1) primary, resulting from insufficient food intake, or (2) secondary, resulting from impairment in the absorption, transportation, or utilization of an adequate food intake. A secondary nutritional deficiency is not a result of inadequate dietary intake but of some factor that prevents the body from utilizing or absorbing ingested food and thus causes a depletion of nutrients necessary for the tissues. Nevertheless, many dentists attempt to treat periodontal disorders by prescribing mineral supplements, various members of the vitamin B complex, or vitamin C, with little concern for the true origin of the nutritional inadequacy, or with no evidence that a deficiency exists. Indiscriminate use of doses of high potency vitamin C to treat "bleeding gums," reduce periodontal pockets, or accelerate regeneration in wound healing has not produced the clinical results claimed by those who advocate this form of therapy.

Tissue Changes Related to Nutritional Deficiencies

To describe the changes one may expect to find in periodontal tissues as a result of nutritional deficiencies, the various food classes will be discussed in relation to their nutrient qualities.

1. *Protein.* Animal experiments have shown protein deficiency to be a factor in postoperative wound healing and a causative factor in periodontal disturbances. Degeneration of connective tissue in the gingiva and periodontal ligament, as well as osteoporosis of the alveolar bone and retarded deposition of cementum, has been shown to occur in rats. Proteins are also essential in combating infection. A prolonged protein deficiency may lead to marked depletion of bone marrow and lymphoid tissues and of the antibody mechanism's capacity to synthesize antibody protein.

Antibodies and leukocytes are basically protein in nature, being derived from amino acids supplied by dietary protein. Thus, it is possible that a protein deficiency may lower resistance to toxic products produced by bacterial plaque.

2. *Fats and carbohydrates.* The dental literature contains very little information about the relationship of fats and carbohydrates to gingival or periodontal disease. Fats and carbohydrates are essentially energy-yielding foods. If carbohydrates are absent from the diet and fats alone supply all energy requirements, acidosis results. Experiments in rats have shown that, if fats are absent from the diet and energy requirements are supplied by carbohydrates alone, deficiency diseases develop. This deficiency is caused by a lack of highly unsaturated fatty acids.

Some evidence suggests that capillary resistance is considerably decreased in human subjects when the diet is moderately low in fats. Since capillary resistance and the normal functional physiology of the periodontium are intimately related, the importance of fats in the diet is apparent. Fats also appear to be related to the normal metabolism of certain minerals.

3. *Minerals*. Many experiments have dealt with the various minerals and their relationship to the periodontal tissues. Periodontal disturbances, particularly of bone, were readily produced in laboratory animals maintained on diets that were low in calcium and phosphorus or that had an improper balance of these minerals.

Studies have shown deviations from normal ranges in a greater percentage of patients with periodontitis than in a control group free from periodontal disease. This was the case for deficiencies in calcium, alkaline phosphatase, and other blood factors.

4. *Vitamins*. Vitamins are accessory food substances and are components of enzyme systems essential to cell and tissue health. One characteristic of vitamins is that they are interdependent and interrelated. For example, lack of vitamin C affects the metabolism of vitamin B; proper amounts of vitamin C and calcium improve the utilization of iron; and the presence of salicylates interferes with the formation of vitamin K. It has been demonstrated that certain oral lesions, formerly attributed to a specific vitamin deficiency, may actually be caused by lack of any one of several vitamins. Atrophic lingual glossitis or cheilosis, for example, may respond to treatment with iron, riboflavin, or pyridoxine, or a combination of all three.

Evaluating the Nutrition

The first step in evaluating a patient's nutritional status is to obtain a dietary history listing all foodstuffs eaten for 7 consecutive days. At most health centers, it is also possible to obtain a computerized analysis of the patient's dietary status. Any deficiencies noted would be considered primary nutritional deficiencies.

The second step is to discover any systemic factors that may influence the utilization and absorption of nutrients. Gastrointestinal disease, loss of appetite because of infection, food allergy, diarrhea, gallbladder disease, diabetes, thyroid disease, adrenal dysfunction, etc.—all may contribute to a nutritional deficiency. The inactivation of vitamins such as thiamine or ascorbic acid occurs in achlorhydria; excessive amounts of oxalic acid (spinach) prevent utilization of minerals by the body; polyuria associated with diabetes or fever affects the nitrogen retention of the tissues as well as the water balance of the body; sprue, ulcerative colitis, dysentery, and other diseases that interfere with fat absorption, inhibit the absorption of the fat-soluble vitamins A, D, E, and K. These may be considered secondary factors if an adequate diet has been maintained.

A third step in nutritional evaluation is clinical examination of the patient for signs or symptoms suggestive of deficiencies. General weakness, fatigue, poor appetite, sore lips, sore mouth or tongue, diarrhea, or photophobia are just a few of the many signs that may suggest a nutritional deficiency.

A fourth step that may be employed is the use of pertinent laboratory examinations of the blood and urine. From a practical point of view, the laboratory procedures are seldom used.

In the presence of serious systemic disease, the dentist should refer the patient to a physician for dietary counseling and therapy. In general, whenever nutritional inadequacies are suspected, the patient's physician should be consulted.

4 Periodontal disease and pocket formation

Gingivitis
- Definition
- Early signs
- Differential diagnosis
- Extension of gingivitis

Periodontitis
- Definition
- Pocket formation
- Classification of pockets
- Spread of inflammation
- Horizontal vs. vertical bone loss
- Etiology of the infrabony pocket
- Bifurcation or trifurcation involvement
- Symptoms of periodontitis
- Clinical and histologic features of periodontitis

Juvenile periodontitis (Periodontosis)

Gingivitis and periodontitis are the two most common diseases of the periodontium, perhaps the most common diseases known to mankind. These two diseases constitute the major therapeutic challenge to the general practitioner and the specialist alike. The more one understands about the pathogenesis of periodontal disease, the better able he will be to arrest or reverse the disease process.

Gingivitis

DEFINITION

Gingivitis is the general term applied to inflammation of the gingiva. It is usually the result of injury by microorganisms and their products, and it may be either an acute or a chronic process, the latter being more common. The first line of defense against injury of the gingiva is the sulcular epithelium. Once this is broken, only the resistance of the individual or the removal of the etiologic agent(s) can prevent a continuing sequence of events that may lead to advanced periodontal disease and eventual loss of the natural dentition.

EARLY SIGNS

Gingivitis most frequently begins in the area of the col. One of the earliest signs of the disease is bleeding, quickly followed by alterations in form and color. The following tabulation summarizes clinical and related histopathologic changes that occur in gingivitis.

Clinical change	Underlying histologic changes
1. Gingival bleeding	Ulceration of the sulcular epithelium, with engorged capillaries extending near the surface
2. Redness	Hyperemia, with dilatation and engorgement of capillaries
3. Swelling, puffiness	Infiltration of connective tissues by fluid and cells of inflammatory exudate
4. Loss of gingival tone	Inflammation, with destruction of gingival fiber apparatus
5. Loss of stippling	Edema in underlying connective tissue
6. Firm, leathery consistency	Fibrosis associated with long-standing chronic inflammation
7. Gingival pocket formation	Inflammation, with ulceration of sulcular epithelium and gingival enlargement

DIFFERENTIAL DIAGNOSIS

There are various types of gingivitis. Abrasions, burns, allergic manifestations, necrotizing ulcerative gingivitis, herpetic gingivostomatitis, and gingivitis exaggerated by leukemia, hormonal imbalance, or other systemic disease are some of the conditions that must be considered when gingival inflammation is observed.

EXTENSION OF GINGIVITIS

It should be emphasized that, in the vast majority of cases, gingivitis is a bacterial infection resulting from inadequate plaque control. If the injurious agents are removed, it is highly probable that the tissue will repair and return to normal. If they are not removed, however, the inflammation and infective products of the bacteria will usually spread to the attachment apparatus.

Periodontitis

DEFINITION

Once the inflammatory process involves the alveolar crest, the disease is called periodontitis. The periodontal fibers immediately apical to the sulcus are disrupted, and the junctional epithelium migrates along the root surface, resulting in a deeper sulcus (pocket).

POCKET FORMATION

A pocket is a gingival sulcus pathologically deepened by periodontal disease. It is bordered by the tooth on one side, by ulcerated epithelium on the other, and has the junctional epithelium as its base. Deepening of the sulcus can occur in three ways: (1) By movement of the free gingival margin coronally, as observed in gingivitis; (2) by movement of the junctional epithelium apically, with separation of the coronal portion from the tooth; and (3) by a combination of (1) and (2) (fig. 4-1).

CLASSIFICATION OF POCKETS

Pockets may be classified as follows (fig. 4-2):

1. *Gingival pocket:* deepening of the gingival sulcus, mainly owing to an increase in the size of the gingiva, without any appreciable loss of the underlying tissues or apical migration of the junctional epithelium (fig. 4-2a).

2. *Suprabony pocket:* deepening of the gingival sulcus, with destruction of the adjacent periodontal ligament and alveolar bone, associated with apical migration of the junctional epithelium. The bottom of the pocket and the junctional epithelium always remain coronal to the alveolar bone (fig. 4-2b).

3. *Infrabony (intrabony) pocket:* deepening of the gingival sulcus to a level where the bottom of the pocket and the junctional epithelium are apical to the crest of the alveolar bone. In this type of pocket, the alveolar bone becomes a part of the pocket wall. One, two, or three osse-

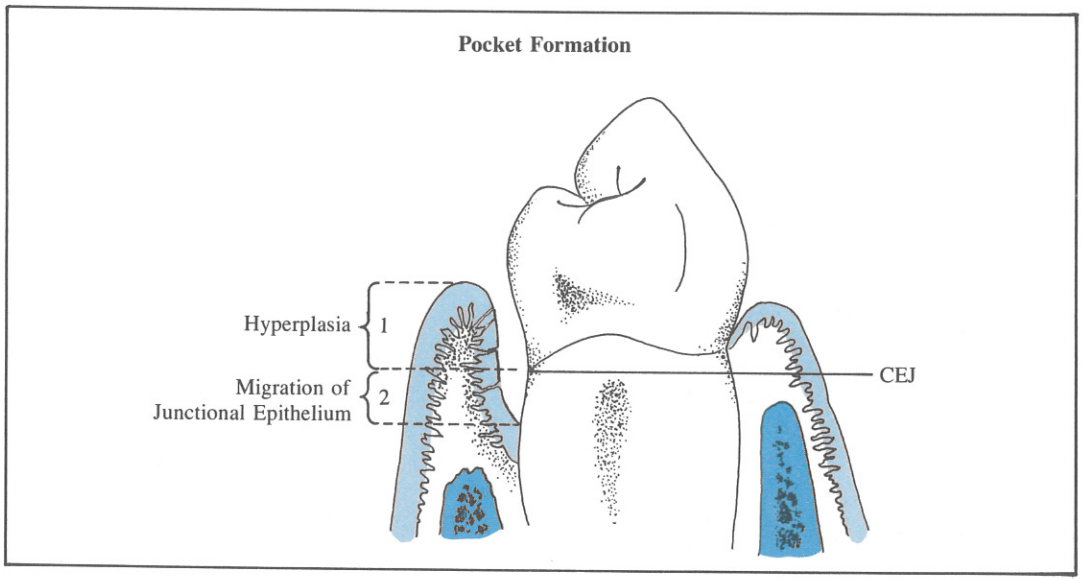


Figure 4-1

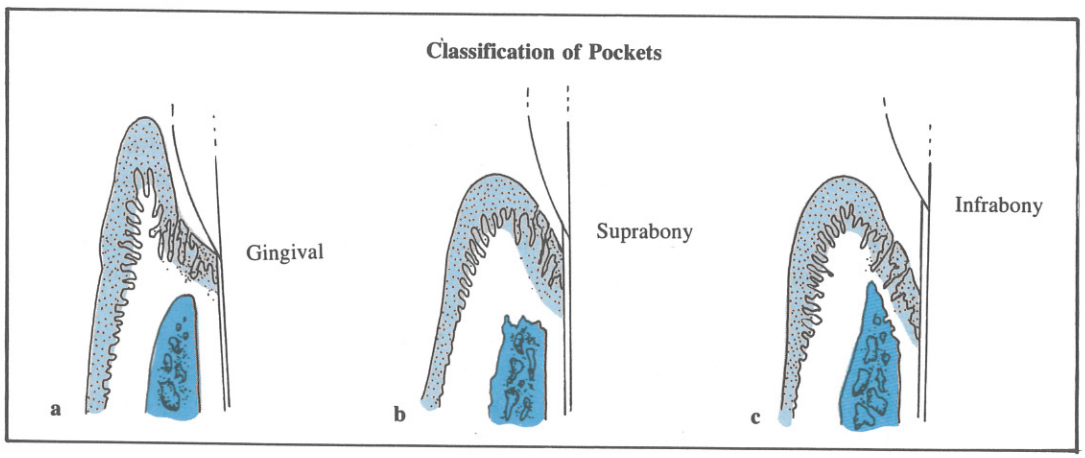


Figure 4-2

ous walls, or various combinations thereof, may remain, depending on the amount and pattern of bone loss (fig. 4-2c).

SPREAD OF INFLAMMATION

Periodontitis usually develops as a sequel to persistent chronic gingivitis and has an identical etiology. Interdentally, inflammation and bacterial products spread from the gingiva to the alveolar process along the neurovascular bundle of the interdental canal at the crest of the septum (fig. 4-3a(1)). Inflammation spreads along the course of the vascular channels because the loose connective tissue surrounding the neurovascular bundles offers less resistance than the dense fibers of the periodontal ligament. The location where the inflammation enters the bone depends on the location of the vessels. In some instances, large vessels will exit at one side of the alveolar crest, permitting direct spread of inflammation into the marginal portion of the periodontal ligament (fig. 4-3a(1a)). After reaching the marrow spaces, the destructive process extends laterally into the periodontal ligament via the intra-alveolar openings (fig. 4-3a(2)). On the facial and lingual

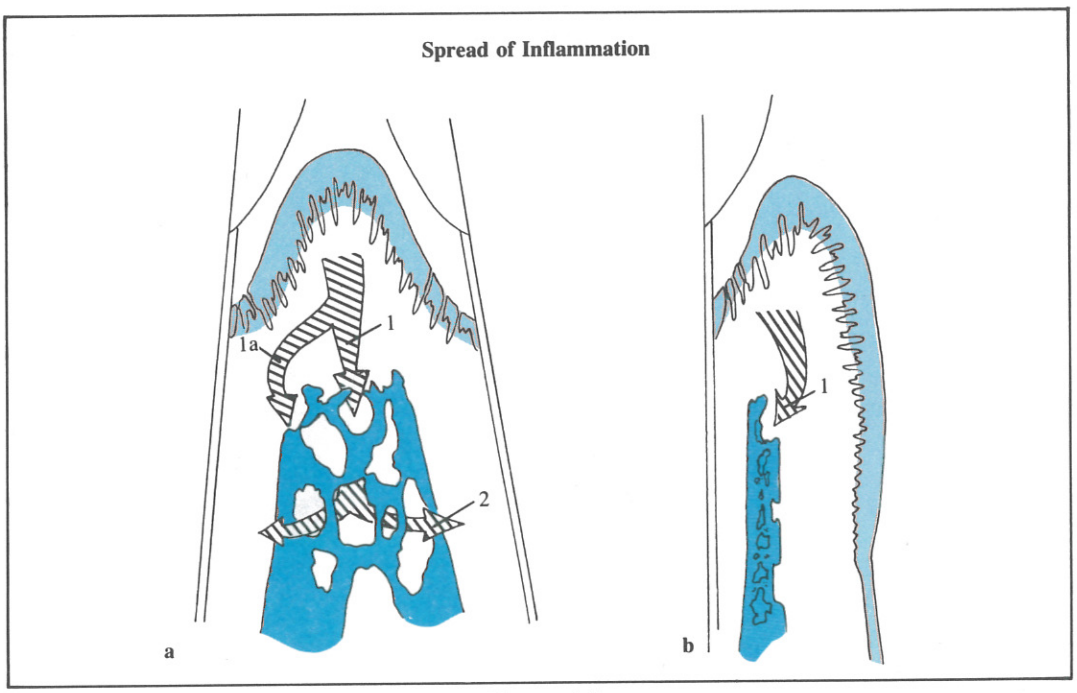


Figure 4-3

surfaces, the destructive process spreads along the supraperiosteal vessels and penetrates into the marrow spaces via the channels in the outer cortex (fig. 4-3b(1)).

Extension of the chronic inflammatory process into the alveolar bone is marked by infiltration of the marrow by leukocytes, new blood vessels, and proliferating fibroblasts. There is marked osteoclastic activity. Progressive extension is accompanied by destruction of the trabeculae and subsequent reduction in the height of the alveolar bone. This destruction is not a continuous process. It is accompanied by osteoblastic activity and new bone formation even in the presence of inflammation. Likewise, there is constant re-formation of the transseptal fibers as the attachment apparatus is destroyed. Alveolar bone loss does not occur until the physiologic equilibrium of bone is disturbed to the point where resorption exceeds formation. The general resistance factors of the individual play an important role in governing the rate at which bone loss progresses in untreated periodontal disease.

HORIZONTAL VS. VERTICAL BONE LOSS

Horizontal bone loss occurs when there is an overall reduction in height of the alveolar crest. Suprabony pockets form when the epithelial attachment migrates apically but remains coronal to the alveolar crest (fig. 4-2(b)). Vertical bone loss occurs when the inflammatory process extends to the periodontal ligament apical to the crestal bone. Infrabony pockets form when there is downgrowth of the epithelial attachment to a point where the attachment is apical to the alveolar crest (fig. 4-2(c)).

ETIOLOGY OF THE INFRABONY POCKET

Both suprabony and infrabony pockets are the result of plaque infection; however, there is some difference of opinion as to the factors that influence the formation of the infrabony pocket. Most agree that vertical bone loss and subsequent infrabony pocket formation can occur any time there is direct extension of inflammation into the periodontal ligament, in the presence of a sufficient thickness of bone. The controversy arises as to *what* factors alter the pathway of inflammation from crestal bone to the periodontal ligament space. The etiologic mechanisms that have been proposed are as follows:

1. Large vessels that exit on one side of the alveolus.

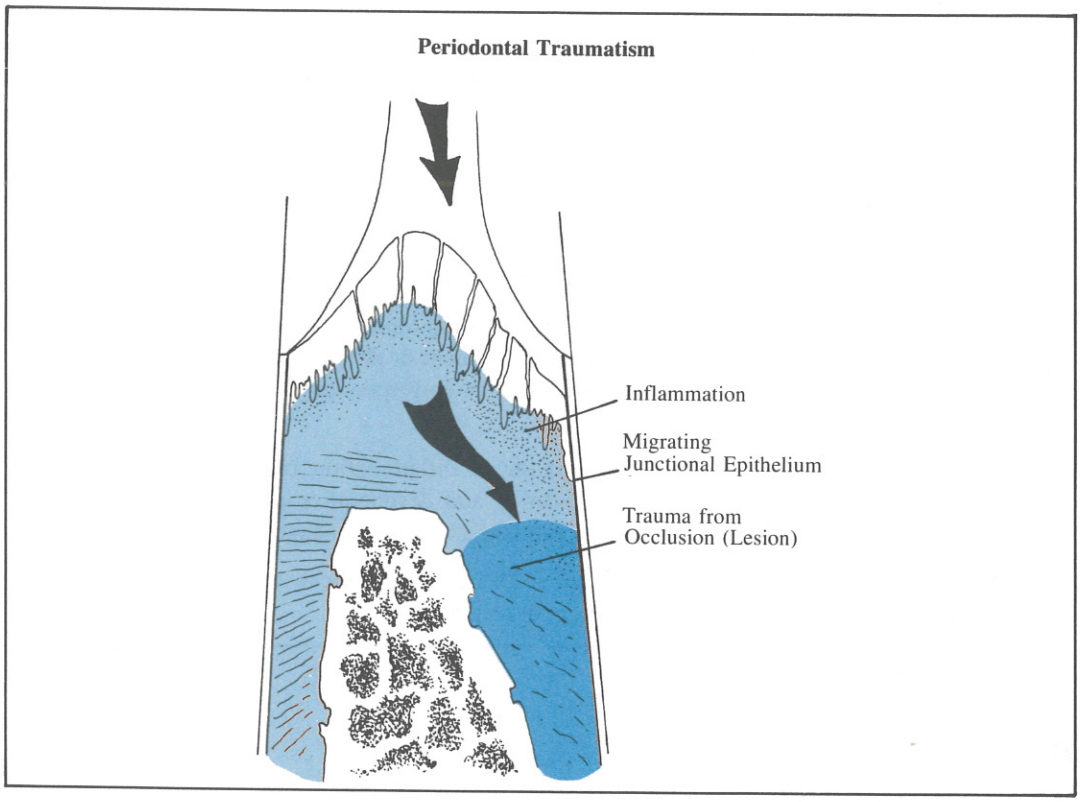


Figure 4-4

2. The forceful wedging of food into the interproximal region may result in unilateral destruction of the attachment apparatus and downgrowth of the epithelial attachment.

3. Periodontal traumatism can produce crestal damage of the periodontal ligament (trauma from occlusion) which, in the presence of an existing inflammation, can result in the migration of the junctional epithelium into the area of destruction (fig. 4-4).

BIFURCATION OR TRIFURCATION INVOLVEMENT

Once the pocket is well established, the chronic inflammatory process is self-perpetuating because the patient is unable to eliminate bacterial colonies from within the pocket. Consequently, destruction continues both in the bone and along the lateral wall of the tooth. Epithelium continues to migrate along the cementum and frequently extends into the interradicular areas of multirooted teeth. This is specifically termed ''bifurcation involvement'' or ''trifurcation involvement,'' or more commonly ''furcation involvement.''

SYMPTOMS OF PERIODONTITIS

Pockets are usually asymptomatic, but patients occasionally complain of a ''deep, gnawing pain,'' itchiness of the gums, sensitivity to heat and cold, a foul taste, bleeding gums, food sticking between the teeth, a toothache in the absence of caries, and increased spacing between their anterior teeth. In spite of a variety of signs and symptoms, or lack thereof, the only reliable method of detecting a pocket's existence is by probing the space between the gingiva and tooth surface.

CLINICAL AND HISTOLOGIC FEATURES OF PERIODONTITIS

The following tabulation summarizes the clinical and histopathologic features of the suprabony and infrabony pocket.

Clinical feature	Histologic feature
1. Bleeding and pain on probing	Ulceration of the sulcular epithelium
2. Bluish-red discoloration of the gingiva	Circulatory stagnation of chronic inflammation
3. Smooth, shiny gingival surface, with loss of stippling	Atrophy of the epithelium and edema in connective tissues
4. Flaccidity of gingiva	Destruction of the gingival fiber apparatus
5. Exposed root surfaces	Result of long-standing chronic inflammation, with progressive apical movement of the junctional epithelium and the gingival margin, with corresponding loss of the alveolar process
6. Occasionally, pink, firm, heavily stippled gingiva, with deep pocket formation	Reparative phase of inflammation (fibrosis) predominating over the exudative and degenerative phase; the pocket wall, however, presents degenerative changes and ulceration
7. Suppuration	Ulceration of epithelium, *not* indicative of severity of disease process or depth of pocket

Juvenile Periodontitis (Periodontosis)

There are a variety of opinions as to the nature, prevalence, and pathologic features of this disease. In 1966, the World Workshop in Periodontics felt that the term ''periodontosis'' was ambiguous, that a specific disease entity could not be defined, and that the term should be eliminated. There is evidence, however, that a clinical entity different from adult periodontitis may occur in adolescents and young adults. Hence the terms ''juvenile periodontitis'' or ''rapid destructive juvenile periodontitis'' have been proposed. The disease is characterized by rapid and extensive bone loss, and loosening and migration of the teeth without apparent accountable bacterial or local contributing factors. Recently, one investigator isolated a number of previously unidentified anaerobes from the pockets of patients with juvenile periodontitis. It seems possible, then, that this rare disease can also be of bacterial origin.

5 Diagnosis, prognosis, and treatment planning

- Diagnosis
  - Periodontal chart
  - Health survey
    - Medical history
    - Dental history
  - General dental survey
  - Periodontal survey
  - Occlusal survey
    - Analysis of the occlusion
    - Periodontal traumatism
  - Radiographic survey
  - Deposits survey
- Prognosis
  - Overall prognosis
  - Prognosis of individual teeth
- Treatment planning
  - Objectives of periodontal therapy
  - Order of treatment
    - Initial preparation
    - Pocket elimination
    - Restorative treatment
    - Maintenance

Successful management of periodontal disease depends greatly on the systematic conversion of examination data into a comprehensive, written treatment plan. This is obvious. It has been discussed at length in textbooks, journals, and lectures. Yet all degrees of periodontal destruction are evident in patients who have had constant dental supervision for 30 years. This has been termed "supervised neglect."

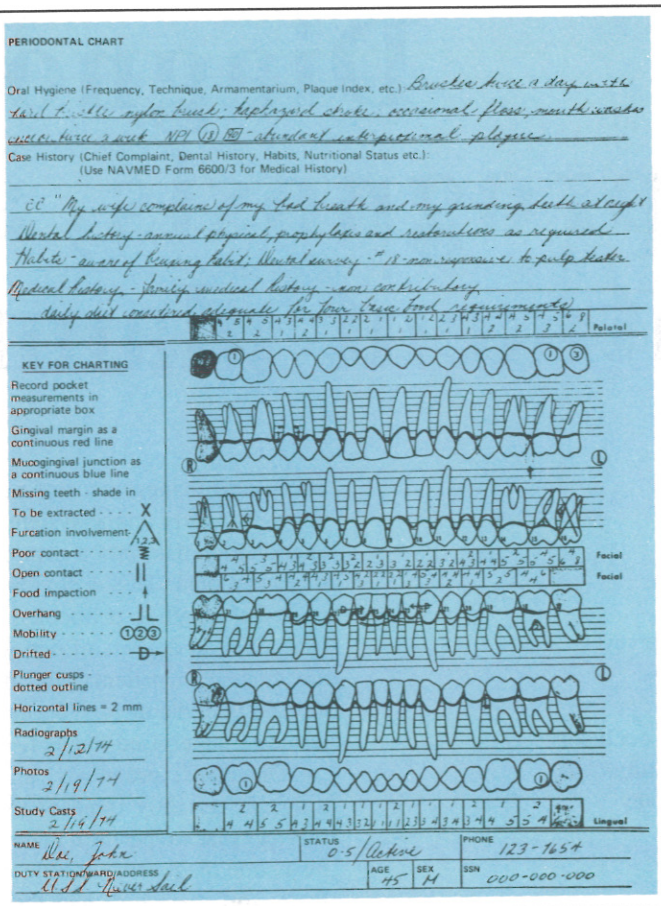
Diagnosis, prognosis, and treatment planning are certainly three of the most important services that we perform in dentistry. In order to be a good diagnostician, one must gather all the facts, sort them out, and then assimilate them into a step-by-step road map or treatment plan. The meticulous manner in which the clinician approaches his fact-gathering appointment(s) will determine success or failure in case management.

This chapter is designed to serve as a guide for the gathering of information and to discuss its use in the formation of a prognosis and treatment plan.

Diagnosis

PERIODONTAL CHART

The practitioner's attack on periodontal diseases will be more productive and less frustrating if information is recorded on a good form, one that serves as a fact-gathering guide, allows brief shorthand notations, and provides space in which to formulate the treatment plan. The charts in figure 5-1 **a** and **b** serve this purpose.


PERIODONTAL CHART

Oral Hygiene (Frequency, Technique, Armamentarium, Plaque Index, etc.): Brushes twice a day with hard t. Uses nylon brush; haphazard stroke. occasional floss, mouth rinses once or twice a week. NPI (3) (50) - abundant interproximal plaque.

Case History (Chief Complaint, Dental History, Habits, Nutritional Status etc.):
(Use NAVMED Form 6600/3 for Medical History)

CC "My wife complains of my bad breath and my grinding teeth at night"
Dental history - annual physical, prophylaxis and restorations as required.
Habits - aware of bruxing habit. Mental survey - "B" - non-aggressive to pulp factor.
Medical history - family medical history - non-contributory.
daily diet - considered adequate for true basic food requirements.

KEY FOR CHARTING

Record pocket measurements in appropriate box
Gingival margin as a continuous red line
Mucogingival junction as a continuous blue line
Missing teeth - shade in
To be extracted - X
Furcation involvement - 1 2 3
Poor contact
Open contact
Food impaction
Overhang
Mobility - 1 2 3
Drifted - D
Plunger cusps - dotted outline
Horizontal lines = 2 mm
Radiographs 2/12/74
Photos 2/19/74
Study Casts 2/14/74

Palatal
Facial
Facial
Lingual

NAME: Doe, John | STATUS: 0-5/Active | PHONE: 123-7654
DUTY STATION/WARD/ADDRESS: USS Navy Sail | AGE: 45 | SEX: M | SSN: 000-000-000

Figure 5-1a

The basic data-gathering that precedes periodontal therapy may be considered as a series of surveys. As these surveys are completed, it may be helpful to estimate the comparative influence of each survey area on the patient's condition. The survey areas are:

1. Health survey.
2. General dental survey.
3. Periodontal survey.
4. Occlusal survey.
5. Radiographic survey.
6. Deposits survey.

Stated simply, virtually all periodontal disease is a struggle between the patient's resistance factors and dentobacterial plaque. Every single factor surveyed simply modifies the influence of either the disease agent or the host resistance.

HEALTH SURVEY

The health survey includes both a medical and a dental history.

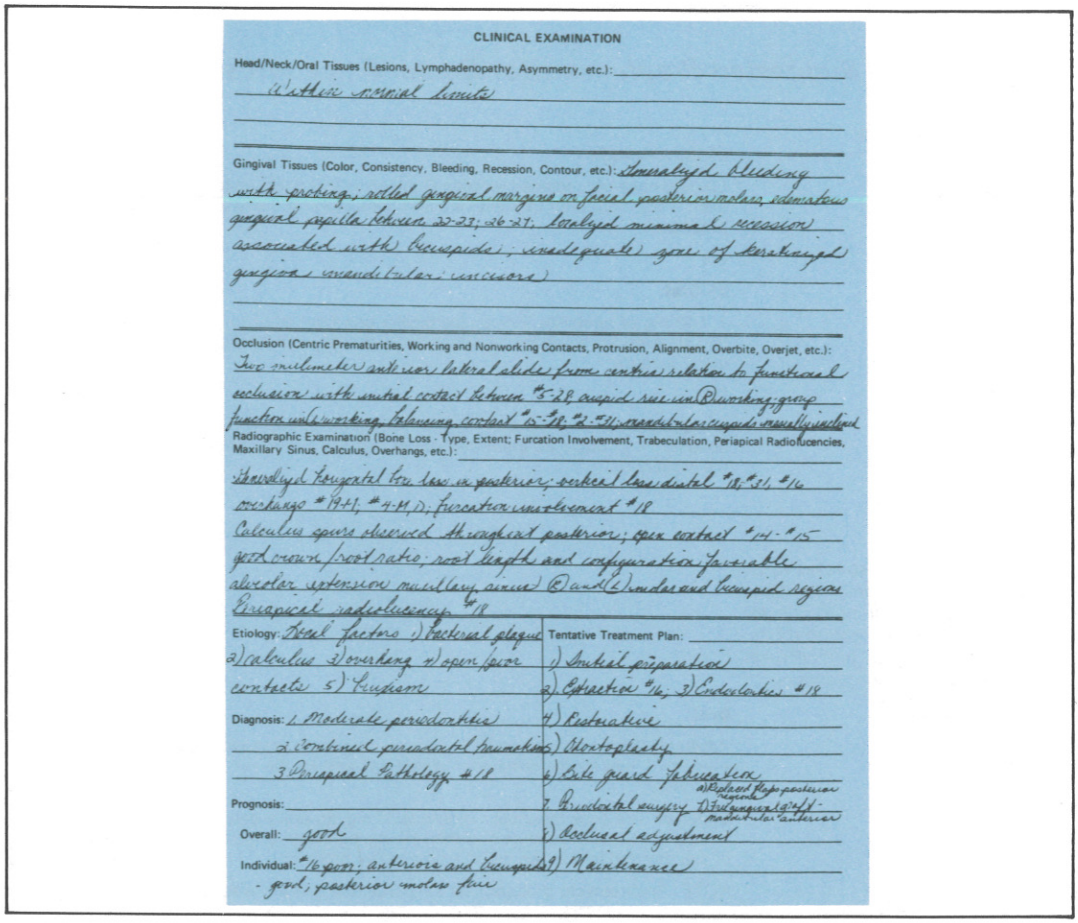
Medical History

The Dental Health Questionnaire, NAVMED 6600/3, which has several questions on medical history pertinent to patient management, serves as an excellent screening examination from which the clinician can expand into questionable areas.

A medical history is vital for three major reasons:

1. Oral manifestations of certain systemic conditions must be noted. These may include leukemia, diabetes mellitus, hormonal disturbances, etc. An alert oral diagnostician, in addition to ensuring good management for his patient, may detect conditions having important health implications.

2. Systemic conditions may alter the tissue response to inflammation. These would include pregnancy, diabetes mellitus, blood dyscrasias, nutritional deficiencies, and hypertensive cardiovascular disease.



CLINICAL EXAMINATION

Head/Neck/Oral Tissues (Lesions, Lymphadenopathy, Asymmetry, etc.):
Within normal limits

Gingival Tissues (Color, Consistency, Bleeding, Recession, Contour, etc.): Generalized bleeding with probing; rolled gingival margins on facial posterior molars, edematous gingival papilla between 27-23; 26-27. Localized minimal recession associated with bicuspids; inadequate zone of keratinized gingiva - mandibular incisors

Occlusion (Centric Prematurities, Working and Nonworking Contacts, Protrusion, Alignment, Overbite, Overjet, etc.):
Two millimeter anterior lateral slide from centric relation to functional occlusion with mutual contact between #5-28 cuspid rise in (R) working group function w/a working, balancing contact #5-28, #2-#11 mandibular cuspids generally inclined

Radiographic Examination (Bone Loss - Type, Extent; Furcation Involvement, Trabeculation, Periapical Radiolucencies, Maxillary Sinus, Calculus, Overhangs, etc.):
Generalized horizontal bone loss in posterior; vertical bone distal #18, #31, #16 overhangs #19+1, #4-M, D; furcation involvement #18
Calculus spurs observed throughout posterior; open contact #14-#15
good crown/root ratio; root length and configuration favorable
alveolar extension maxillary sinus (R) and (L) molar and bicuspid region
Periapical radiolucency #18

Etiology: Local factors 1) Bacterial plaque 2) Calculus 3) Overhang 4) open facial contacts 5) Trauma

Diagnosis: 1 Moderate periodontitis
2 Combined periodontal pathology
3 Periapical Pathology #18

Prognosis:
Overall: good
Individual: #16 poor; anterior and bicuspids - good, posterior molars fair

Tentative Treatment Plan:
1) Initial preparation
2) Extraction #16, 3) Endodontics #18
4) Restorative
5) Mucoplasty
6) Bite guard fabrication
7) Periodontal surgery (R) Widman type flap posterior regions (R) modified Widman anterior
8) Occlusal adjustment
9) Maintenance

Figure 5-1b

3. Certain systemic conditions require modification of both primary and supportive periodontal therapy. These include allergic conditions, rheumatic fever syndrome, diabetes mellitus, endocrine disorders, cardiovascular diseases and valvular prostheses, drug therapy (endocrine, corticosteroid, anticoagulant), and psychological problems.

For example, it is imperative that the therapist recognize patients with valvular damage or valvular prostheses immediately. Any probing or manipulation of the gingival tissue (periodontal examination, scaling, curettage, root canal treatment, extractions, periodontal surgery, etc.) results in a bacteremia which could result in subacute bacterial endocarditis.

Patients who have cardiovascular problems, such as rheumatic heart disease, congenital heart disease, prosthetic valves, etc., should be managed in coordination with a medical officer, preferably a cardiologist. Consultation forms (SF 513) confirming the presence or absence of disorders should be retained in the patient's dental folder. Occasionally, patients who claim a history of rheumatic fever are reported by the cardiologist to be free from valvular damage or associated sequelae, thereby negating the need for antibiotic coverage. It is extremely important, however, that all patients with a history of rheumatic fever be carefully evaluated by a physician prior to *any* dental treatment. Written consultation—not telephoned—is mandatory.

The patient with an *established* history of rheumatic fever or congenital heart disease should be given medication according to the following recommendations of the American Heart Association.*

*From "Prevention of Bacterial Endocarditis" © 1965, 1972 American Heart Association. Reprinted with permission. Leaflet.

Suggested Prophylaxis for Dental Procedures

I. For most patients:

Penicillin

a) Intramuscular:

600,000 units of procaine penicillin G mixed with 200,000 units of crystalline penicillin G one hour prior to procedure and once daily for the two days* following the procedure.

OR

b) Oral:

1. 500 mg of penicillin V or phenethicillin one hour prior to procedure and then 250 mg every six hours for the remainder of that day and for the two days* following the procedure.

OR

2. 1,200,000 units of penicillin G one hour prior to procedure and then 600,000 units every six hours for the remainder of that day and for the two days* following the procedure.

II. For patients suspected to be allergic to penicillin or for those on continual *oral* penicillin for rheumatic fever prophylaxis, who may harbor penicillin-resistant viridans streptococci:

Erythromycin

Oral

Adults: 500 mg one and one-half to two hours prior to procedure and then 250 mg every six hours for the remainder of that day and for the two days* following the procedure.

Children: The dose for small children is 20 mg/kg orally one and one-half to two hours prior to the procedure and then 10 mg/kg every six hours for the remainder of that day and for the two days* following the procedure.

NOTE: Erythromycin preparations for parenteral use are also available.

*Or longer in the case of delayed healing.

The drug requirements for the patient with a heart prosthesis differ from the foregoing regimen, and treatment on an inpatient basis is ordinarily required. If the patient must be treated as an outpatient, the following regimen may be instituted:†

Ampicillin, 0.50 Gm orally every 6 hours, beginning 2 hours before procedure and continue for 3 days

Kanamycin, 0.50 Gm intramuscularly 1 hour before procedure

Kanamycin, 0.25 Gm intramuscularly 4 hours after procedure

If kidney function is impaired, the dosage of kanamycin is limited to the initial 0.50 Gm intramuscularly 1 hour before procedure.

Patients with cardiovascular problems are only one example of the importance of a thorough medical history. The often repeated phrase "never treat a stranger" is good advice.

Dental History

Before the intraoral examination, a complete dental history should be obtained. This provides an opportunity to assay the patient's attitude and establish rapport, in addition to learning of past dental disease, response, and treatment. It is likewise important to determine what methods of tooth cleansing the patient is presently using and his general dental I.Q.

†Bottomley, W. K., Willis, P. W., III, and Fekety, R. F. The dental patient with a heart valve prosthesis: Preoperative and postoperative consideration, J. Mich. Dent. Assoc. 51:149–151 April 1969.

GENERAL DENTAL SURVEY

The overall impression gained here will begin to establish the magnitude of the problem. The following points should be observed and noted:

1. Soft tissue survey. This is the oral cancer search. Other lesions may be noted but few have as severe consequences, especially if not detected early or if completely overlooked.
2. Arch alinement, morphologic malocclusion, and migration of teeth.
3. Caries. Location, type, and extent.
4. Restorative dentistry. Adequacy of restorations and prostheses. These must be viewed in relation to plaque retention, prevention of plaque removal, traumatogenic occlusion, and excessive leverage of torquing forces.
5. Habits. Smoking, tongue-thrusting, bruxism, clenching, etc.
6. Pulpal status of all teeth should be determined, insofar as possible. The relationships between pulpal status and periodontal disease have become increasingly appreciated and may alter treatment planning.
7. Mobility of teeth is a critical diagnostic and prognostic consideration. Some mobility is normal and may vary during the day, according to diet and stress. Pathologic mobility has several principal causes:
    a. Gingival and periodontal inflammation.
    b. Parafunctional occlusal habits (bruxism, clenching, etc.).
    c. Occlusal prematurities.
    d. Loss of supporting bone.
    e. Traumatic torquing forces applied to clasped teeth by removable partial dentures.

In addition, some transient mobility may follow periodontal therapy, endodontic therapy, and traumatic injuries.

Tooth movement is measured by applying force buccolingually between two dental instrument handles. Mobility is usually graded as 1, 2, or 3 (fig. 5-2). Grade 1 represents the first distinguishable sign of movement greater than normal; Grade 2 is recorded if there is movement of at least 1 mm; Grade 3 is recorded if the tooth moves more than 1 mm in any direction and/or is depressible.

Elimination or control of pathologic tooth mobility is gained through interference with the causative factors.

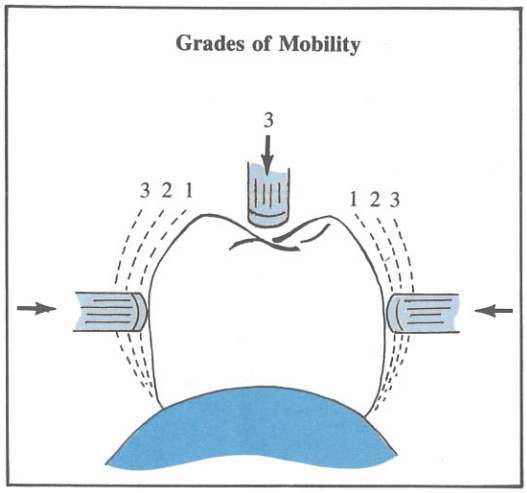


Figure 5-2

PERIODONTAL SURVEY

Obviously, this is a very critical part of the diagnostic process. A calibrated periodontal probe, a cowhorn or pigtail explorer, a front reflective surface examining mirror, adequate light, palpation, and air blast must all be employed to supplement visual examination of the periodontal tissues. Figure 5-1 demonstrates how information obtained from a periodontal survey is recorded.

1. Gingival color, form, and consistency are observed and recorded.
2. Bleeding and purulent exudate are immediate indications of disease activity and should be noted. Exudation may be spontaneous or be evident only on probing or palpation. Suppuration is not an indicator of the severity of the disease but merely signifies ulceration of the epithelial wall of the pocket, with a corresponding reaction of the body to infection.
3. Pocket depth is measured from the gingival margin on all teeth with the aid of a calibrated probe. The instrument is held as close to the tooth surface as possible, and gently inserted into the sulcus or pocket until resistance is met. Any bleeding or suppuration is recorded. The probe is walked along the tooth surface, keeping it parallel to the long axis of the tooth. Three measurements are recorded on both the facial and lingual surfaces: The distal, midfacial/midlingual, and mesial line angles (fig. 5-3). When there is heavy calculus formation, it is often impossible to accurately measure pocket depth, since calculus will impede the insertion of the probe. It may be

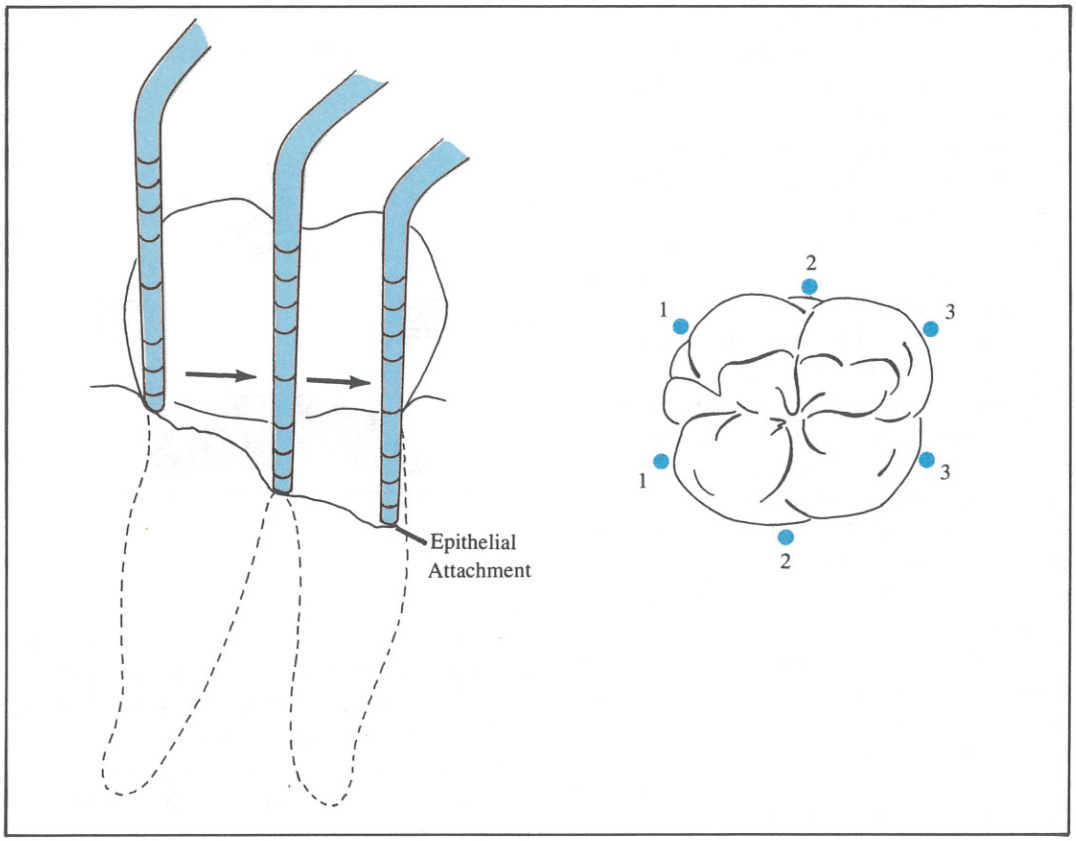


Figure 5-3

necessary then to perform a gross debridement prior to pocket measurement.

4. The relationship of the gingival margin to the cementoenamel junction (recession) is recorded as a continuous line on a chart. If this step is neglected, pocket measurements will be meaningless. A 3 mm pocket, for example, on a tooth with 5 mm of gingival recession, would signify greater destruction of the attachment apparatus than a 5 mm pocket on a tooth with hyperplastic gingiva (fig. 5-4).

5. The general width of keratinized gingiva, the relationship of pocket depth to the mucogingival junction, and the influence of various frenal and muscle attachments on the gingival margin are observed and recorded. These may present special surgical problems.

6. Pathologic invasion of the furcation areas should be determined by careful probing with a curved explorer. The complicated anatomy of these regions presents diagnostic and therapeutic challenges. Furcations are classified as follows (fig. 5-5): Class I (incipient defects), Class II (moderate involvement), and Class III (through-and-through communication). These classes may be defined in greater detail as follows:

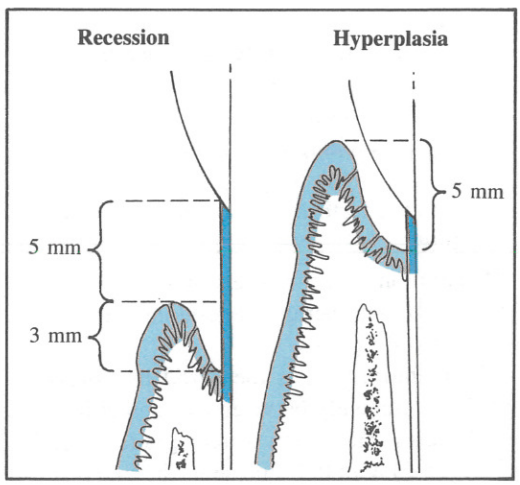


Figure 5-4

Class I: A soft tissue lesion extending to the furcation level but with minimal osseous destruc-

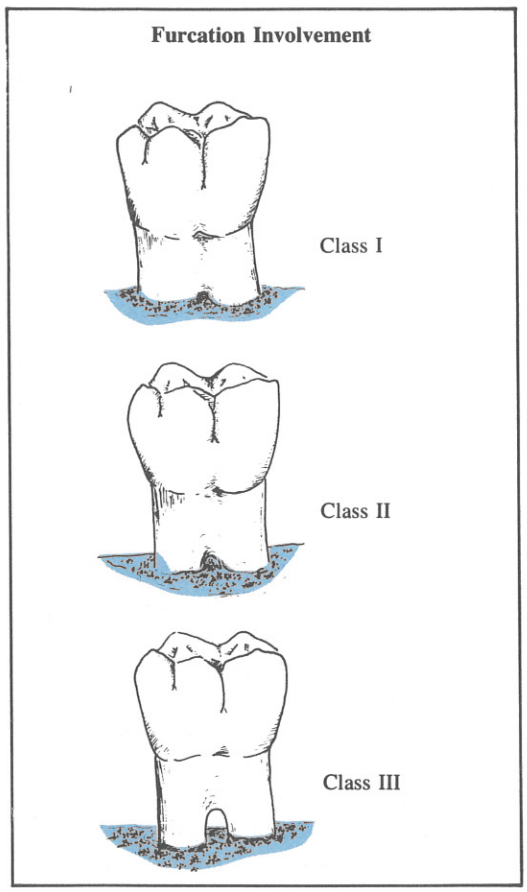


Figure 5-5

tion. Radiographs of these incipient lesions reveal little, if any, evidence of pathology.

Class II: A soft tissue lesion combined with bone loss that permits a probe to enter the furcation from one aspect but not to pass completely through the furcation. This class is further subdivided as follows:

Class II-F: Entry is from the facial aspect only.
Class II-L: Entry is from the lingual aspect only.
Class II-M: Entry is made from the mesial aspect only.
Class II-D: Entry is from the distal aspect only.

Some evidence of Class II involvement can usually be observed in radiographs of mandibular molars, but such evidence may be obscured in radiographs of maxillary molars.

Class III: Lesions with extensive osseous destruction that permits through-and-through passage of the probe. The through-and-through nature of the communication may be obscured by soft tissue.

OCCLUSAL SURVEY

Since occlusion may influence the progress and severity of periodontal disease, the occlusal status of every patient should be evaluated. This approach will enable the therapist to gauge the relative importance of occlusion in each individual case and to determine whether occlusal adjustment is indicated.

Analysis of the Occlusion

The following information must be obtained before a final evaluation of the role of occlusion can be made.

History.

1. Determine parafunctional habits (bruxing, clenching, doodling, fingernail biting, etc.).
2. Determine pain in region of temporomandibular joint, muscle spasms, subluxation.
3. Periodontal abscess formation.
4. Fanning of anterior teeth.
5. Hypersensitivity.
6. Food impaction.

Radiographs. Examine radiographs for:

1. Vertical bone loss.
2. Furcation involvement.
3. Widening of periodontal ligament space.
4. Root fractures.
5. Thickening of lamina dura.
6. Hypercementosis.

Occlusion. Ensure that the patient is in a relaxed position. Using the mouth as an articulator, perform the following procedures:

1. Determine deviation of the mandible (right or left) on opening.

2. Establish initial contact in centric relation. Centric relation refers to the most posterior relation of the mandible to the maxillae at the established vertical dimension. This is a reference point from which the occlusion can be adjusted. To find the initial contact in centric relation, place gentle pressure against the center of the mandible and guide the mandible posteriorly

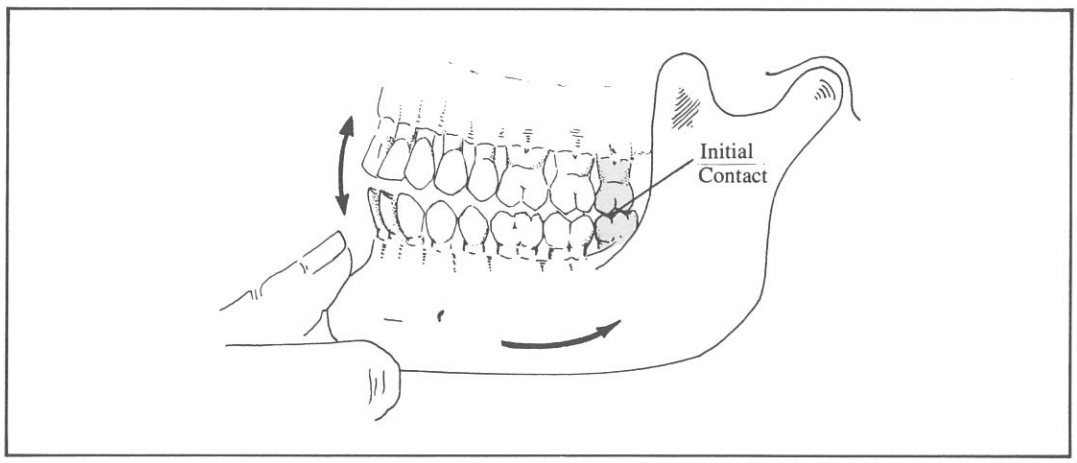


Figure 5-6

(fig. 5-6). Record the first contact in centric relation (first centric prematurity) by tapping the teeth into occlusal indicator wax or articulating ribbon.

3. Determine anterior slide of mandible. Have patient slowly close teeth together from initial contact in centric relation, and observe anterior or lateral movement of the mandible. Also note facial movement of teeth as jaw closes from centric relation to functional (habitual) occlusion.

4. Determine prematurities in functional occlusion. This is the position in which most functional contacts occur. Prematurities in this position are easily determined by asking the patient to slowly close teeth into indicator wax or ribbon. Body posture and head position will affect functional occlusion, so it is advisable to have the patient upright in the chair (fig. 5-7).

5. Determine working contacts. Working contacts occur when the mandible is moved laterally with the teeth in contact. Such contacts do not regularly occur in chewing and swallowing, as once believed, but frequently occur in bruxing. Working contacts are recorded with the patient in an upright position. The patient is asked to glide his mandible right and left from functional position. Contacts are best recorded with ribbon.

6. Determine nonworking contacts. When the mandible is moved to the right (right working), posterior teeth may contact on the left. These contacts are called nonworking (balancing) contacts. Nonworking contacts are potentially the most damaging to the periodontium. They are recorded by placing ribbon on the nonworking side and having the patient move his mandible into working position. Look for long streaked markings on the teeth.

7. Determine cuspid rise. In lateral and protrusive excursions, the mandibular canines and first premolars will sometimes engage the palatal surface of the maxillary canines and will disocclude the incisors, premolars, and molars. This is easily determined by means of ribbon or indicator wax.

8. Determine contacts in protrusive position. Have the patient bite on the anterior teeth in tip-to-tip relation. Establish simultaneous contact from cuspid to cuspid. Also note any posterior teeth that may contact in protrusive position.

9. Determine protrusive excursion. Place the ribbon between the upper and lower anterior teeth. Have the patient close in functional position and then protrude the mandible until the teeth reach protrusive position. Record the teeth that contact during this excursion.

10. Check movement of teeth during chewing; fremitus. Place the ball of the index finger on one tooth at a time and determine if the tooth moves as the patient glides his mandible into working position.

11. Determine tooth/tooth relationships: Open contacts, irregular contacts, food impaction sites, rough incisal/occlusal surfaces.

Study casts. Look for:

1. Plunger cusps.
2. Wear facets.

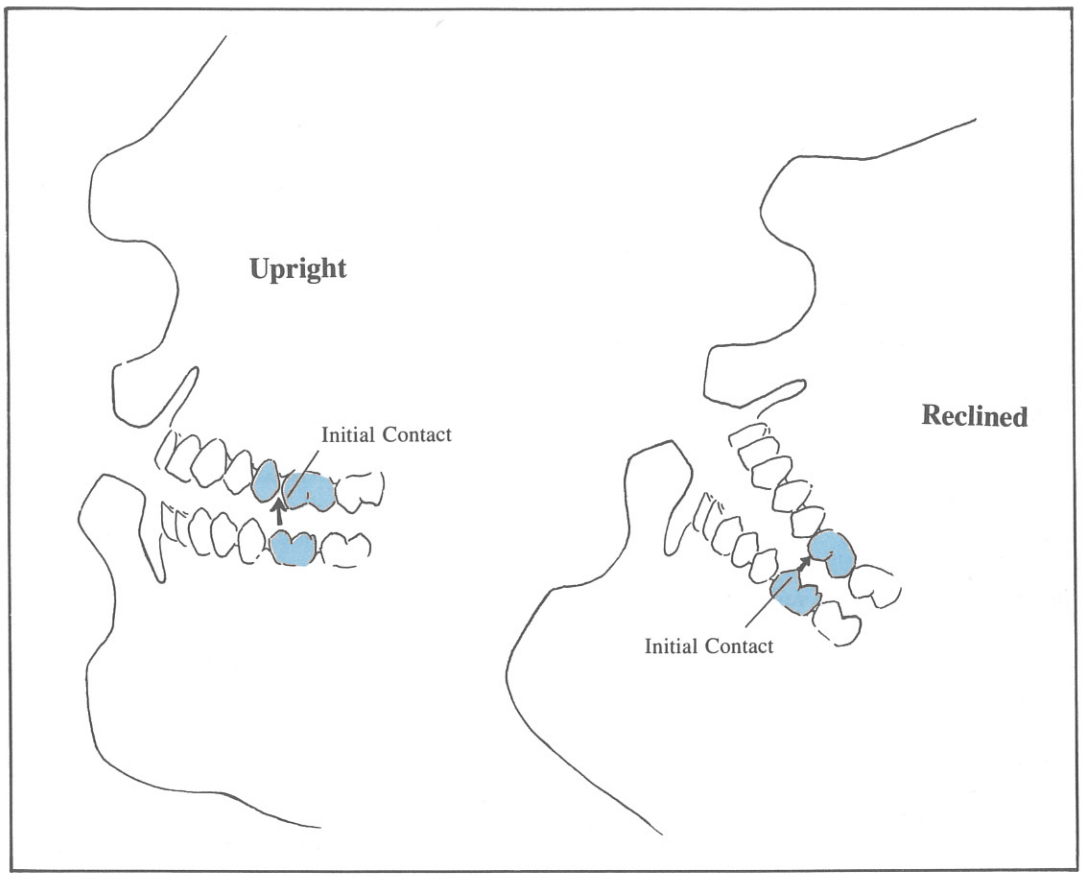


Figure 5-7

3. Malposed teeth.
4. Marginal ridge relationship.
5. Condition of existing restorations (contour, buccolingual dimension).
6. First molar relationship (Angle's classification).
7. Overbite (vertical overlap of teeth).
8. Overjet (horizontal overlap of teeth).

Periodontal Traumatism

Once the occlusal survey is complete, the clinician must relate the data to the existence or nonexistence of periodontal traumatism. The following outline of signs and symptoms will serve as a guide in making the diagnosis:

Clinical signs. Look for:
1. Mobility of teeth.
2. Fremitus.
3. Migration of teeth; "fanning" of anterior teeth.
4. Unusual wear patterns of teeth (facets).
5. Hypertonicity of masticatory muscles.
6. Periodontal abscess formation, especially in deep infrabony defects and furcae.

Symptoms. Periodontal traumatism is often asymptomatic, but the following symptoms may be indicative of this condition:

1. Soreness on percussion and in function is often associated with new restorations and has a short-term history. In chronic periodontal traumatism, pain may be more vague and generalized.
2. Pain and spasm in muscles of mastication.
3. Food impactions due to forceful wedging by opposing teeth.
4. Temporomandibular joint pain/dysfunction syndrome.
5. "Looseness of teeth," vague "itching," and tendency to grind or initiate parafunction on certain teeth.

6. Thermal hypersensitivity of teeth in trauma.

Radiographic signs.

1. Alterations in the lamina dura:
   a. Uneven thickening may be associated with tensional forces but is unreliable.
   b. Severe occlusal force may cause a complete loss of lamina dura.
2. Alteration in the periodontal ligament space. Widening may mean increased function or periodontal traumatism. The widening may be compensatory, especially if the lamina dura is thickened and intact.
3. Root resorption may be due to excessive force in orthodontics, bruxism, or reconstruction therapy.
4. Hypercementosis may be a compensatory phenomenon to increase resistance to occlusal forces.
5. Osteosclerosis may occasionally be observed.
6. Angular bone loss and bone loss in furcation areas have been suggested in association with excessive occlusal force.
7. Root fracture.

Microscopic changes. Various functional conditions, including periodontal traumatism, produce some changes within the periodontium that can be observed microscopically. These changes include:

1. Changes resulting from nonfunction:
   a. Bone marrow spaces widen.
   b. Trabeculae become thin and disoriented.
   c. Periodontal ligament becomes narrow and fibers become disoriented.
2. Changes produced by overfunction (within physiologic limits):
   a. Bone marrow spaces become smaller than normal.
   b. Trabeculae become dense and may achieve a buttressing effect.
   c. Periodontal ligament becomes wider.
3. Changes produced by overfunction on the pressure side (beyond physiologic limits):
   a. Compression of contents of the periodontal ligament.
   b. Hemorrhage and concomitant hematoma:
   c. Thrombosis.
   d. Compression necrosis.
   e. Ischemic necrosis and rupture of vessel walls.
   f. Hyalinization (may occur).
   g. Undermining resorption of the alveolar process, starting from adjacent marrow spaces.
   h. Resorption of cementum.
   i. Root resorption (may occur).
4. Changes produced by overfunction on the tension side (beyond physiologic limits):
   a. Widened periodontal ligament.
   b. Hemorrhage and concomitant hematoma.
   c. Thrombosis and hyalinization (may occur).
   d. Apposition of alveolar process.
   e. Hypercementosis (may occur).
   f. Cemental and periodontal ligament tears (both may occur if occlusal force is of sufficient magnitude).

Prognosis. The accommodative capacity of the periodontium is the key to whether the resultant changes will be damaging. Certain factors may alter the accommodative capacity, including:

1. Age of patient. The accommodative capacity is at its highest in the young patient.
2. Gingival inflammation. The inflammatory process may hasten loss of the alveolar process and enhance the effects of excessive occlusal force on the periodontium.
3. Systemic conditions. These alter tissue responses to occlusal stress with the result that delayed healing occurs and the capacity to withstand forces is lessened. These may include diabetes, hyperparathyroidism, etc.
4. Amount of alveolar process remaining. Loss of supporting bone may sometimes cause a normally physiologic occlusal force to become traumatic. The less remaining bone, the less is the accommodative capacity of the periodontium. This results in an increased crown-to-root ratio.
5. Force.
   a. Direction. Those forces directed *away* from the long axes of the teeth are most detrimental.
   b. Distribution. Forces are more destructive when concentrated on a few teeth than when they are distributed over many pairs.

c. Duration. Forces that are continuous, as in clenching and grinding habits, are potentially more destructive.

d. Frequency. The more frequent the force, the greater the opportunity for damage, e.g., patients swallow with tooth contact 500 to 1,500 times a day. This affords greater frequency of tooth contact than eating meals.

e. Intensity.

The clinician should always be mindful that, regardless of its direction, distribution, duration, frequency, or intensity, what really determines whether a force is traumatic is whether it produces destructive changes in the periodontium or within the stomatognathic system.

RADIOGRAPHIC SURVEY

Radiographs are indispensable aids in the diagnosis of periodontal disease. Radiographic interpretation, however, should be considered along with clinical data in order to establish a final accurate diagnosis. Each diagnostic regimen serves to monitor the accuracy of the other.

General requirements:

1. The radiographic survey should include a full-mouth periapical series, a four-film periodontal bite-wing series, and if facilities permit, panoramic radiographs.
2. Films should be technically adequate in density, contrast, angulation, and inclusion of pertinent anatomic detail.

What radiographs will show (fig. 5-8 depicts many of the features):

1. Root length and morphology.
2. Clinical crown-root ratio.
3. Approximate amount of bone destruction.
4. Relationship of maxillary sinus to periodontal deformity.

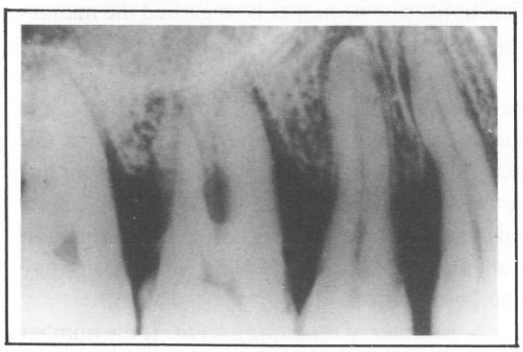


Figure 5-8

5. Condition of interproximal bony crests; horizontal and vertical resorption. It should be noted that the height of normal interseptal bone is usually parallel to a line connecting the cementoenamel junctions of adjacent teeth. When these landmarks are not on the same horizontal plane, the resulting angular appearance of a normal alveolar crest may resemble a pathologic infrabony defect. The astute diagnostician must be aware of these cementoenamel junction relationships, as well as of the dense appearance of healthy crestal lamina dura, to avoid unnecessary periodontal osseous surgery. Crown form and stage of eruption also influence the morphology of interseptal bone.
6. Widening of periodontal ligament space on mesial and distal aspects of the root.
7. Advanced furcation involvements.
8. Periapical pathosis.
9. Calculus.
10. Overhanging restoration.
11. Root fractures.
12. Caries.
13. Root resorption.
14. Cemental tears.

What radiographs will not show:

1. Presence or absence of pockets.
2. Exacting morphology of bone deformities, especially tortuous defects, dehiscences, and fenestration.
3. Tooth mobility.
4. Position and condition of alveolar process on facial and lingual surfaces.
5. Early furcation involvements.
6. Location of epithelial attachment.

Adjunctive devices for radiographic diagnosis. The use of certain radiopaque devices may facilitate periodontal radiographic diagnosis. Calibrated metal Hirschfeld's points inserted prior to radiographic exposure give accurate measurements of pocket depth. Silver endodontic or gutta-percha points may be similarly employed. Fine metal wires may be used to trace sinus tracts and the morphology of tortuous bony defects.

The Everett-Fixott grid may be attached to the film prior to exposure to provide a standardized reference in healing progress of infrabony lesions. Superimposition of the 1 mm grid squares permits more accurate interpretation of osseous regeneration.

a

b

Figure 5-9

DEPOSITS SURVEY

A survey of the existing tooth accumulated materials (TAM) is extremely important. In order to accurately determine the prevalence and distribution of TAM, it is necessary, even to the trained eye, to utilize disclosing solutions, as demonstrated in figures 5-9a and b. For optimal usefulness in monitoring therapeutic progress, these accumulations should be recorded repeatedly on a chart such as the Navy Plaque Index. The Deposits Survey is conducted last because the disclosing media used in this examination mask other important clinical signs, such as changes in gingival coloring. Some clinicians prefer to question patients regarding their current tooth cleansing procedures at this time rather than during the dental history survey. The timing is unimportant so long as the information is obtained that permits the clinician to correlate techniques with effectiveness.

Prognosis

Prognosis is a forecast of the probable response to treatment and the long-term outlook for maintaining a functional dentition. Hopeless cases generally present few problems in establishing an accurate prognosis. Neither do cases of simple gingivitis, which can be expected to respond favorably when local and systemic factors can be controlled. In the borderline cases, however, the decision-making process becomes challenging.

Problems are compounded when the prognosis concerns strategic, severely involved individual teeth on which a large and complex restorative treatment plan often depends. This situation places a heavy burden of responsibility on the diagnostician under any circumstance.

No formula can be established for such situations. Rules of proportional bone loss (such as one-third or one-half of the supporting bone) have been expressed in the literature as condemning a tooth for extraction. In practice, such rules have been of little value. If adhered to rigidly, such rules may lead to the sacrifice of teeth that might have been retained in health. The difficulty with any formula or rule is that there are too many exceptions. The best way of meeting the problem is to establish certain basic principles, criteria of judgment, and probable behavior patterns of doubtful teeth under the conditions in which they must function.

There are two aspects of prognosis: The overall prognosis and the prognosis of individual teeth.

OVERALL PROGNOSIS

Overall prognosis is concerned with the dentition as a whole and is the basic determinant of whether treatment should be undertaken. It includes consideration of the following factors:

1. *Attitude of patient.* The success of periodontal treatment depends primarily on effective daily plaque control. Without patient cooperation—deep personal commitment and involvement in his therapy—prognosis is poor. This holds true no matter how skilled the managing practitioner.

2. *Age of patient.* The younger the patient, the poorer is the prognosis. Given two patients with periodontal involvement of the same degree, it is logical to assume that the younger has far less resistance, because equal damage occurred in a shorter period. It follows that, in a patient with weak resistance, healing and repair may also be impaired.

3. *Number of remaining teeth.* If the number and the distribution of remaining teeth are inade-

quate to support a satisfactory prosthesis, the overall prognosis is poor. Periodontal injury from extensive fixed or removable prostheses constructed on an insufficient number of natural teeth will only hasten bone loss. Inability to establish a satisfactory functional environment for remaining natural teeth diminishes the likelihood of maintaining periodontal health.

4. *Systemic background.* The patient's systemic background affects the overall prognosis in several ways. When extensive periodontal destruction cannot be attributed to local factors, it is reasonable to assume a contributing systemic influence. The detection of systemic factors is usually extremely difficult. For this reason, the prognosis in such patients is usually poor. However, if patients have known systemic disorders that could affect the periodontium (diabetes, nutritional deficiency, hyperthyroidism, hyperparathyroidism, etc.), the prognosis will improve on correction of the disorder.

When periodontal surgery may be compromised because of the patient's health, the prognosis is uncertain. Incapacitating conditions that prevent adequate plaque control by the patient (such as Parkinson's disease) adversely affect the prognosis.

5. *Malocclusion.* Irregular alinement of the teeth, malformation of the jaws, and disturbed occlusal relationships may be important factors in the etiology and the progression of periodontal disease. Correction by orthodontic or prosthetic means is often essential if periodontal treatment is to succeed. The overall prognosis is poor when *relevant* occlusal deformities are not amenable to correction.

6. *Tooth morphology.* The prognosis is poor in patients whose teeth have short, tapered roots and relatively large crowns. The disproportionate crown-root ratio and the reduced root surface available for periodontal support render the periodontium more susceptible to injury by occlusal forces.

7. *Recall availability.* It is increasingly evident that overall long-term prognosis is dependent on the patient's availability and his motivation to seek frequent recall visits, preferably at 3 to 4 month intervals. Since the most highly motivated patients lapse in their efforts at effective plaque control, they need reinforcement and reevaluation at regular intervals. Frequent recall visits also prevent recurrence of the disease process. Patients who are unable to participate in a regular recall system, for whatever reason, are poor risks for extensive periodontal therapy.

PROGNOSIS OF INDIVIDUAL TEETH

This includes consideration of the following factors:

1. *Mobility.* Tooth mobility is caused by one or more of the following: Gingival and periodontal inflammation, parafunctional habits, occlusal prematurities, torquing forces, and loss of supporting bone. Mobility is usually correctable, *unless* the mobility results solely from loss of the attachment apparatus. This is not likely to be corrected. The likelihood of restoring tooth stability is inversely related, then, to the extent to which it is caused by loss of the attachment apparatus.

2. *Teeth adjacent to edentulous areas.* Abutment teeth are subjected to increased functional demands. More rigid standards are required in evaluating the prognosis of teeth in such locations. An immobile tooth should never be made mobile.

3. *Location of remaining bone in relation to individual root surfaces.* When extensive bone loss has occurred on only one root surface, the tooth's center of rotation is more coronal than if all root surfaces were extensively involved (fig. 5-10). Thus, leverage on the periodontium will be more favorable than the extensive bone loss on the single surface suggests.

4. *Relation to adjacent teeth.* When a toqth has a questionable prognosis, the chances of successful treatment should be weighed against the benefits that would accrue to adjacent teeth if that tooth were extracted. Unsuccessful attempts at treatment often jeopardize adjacent teeth. Simple extraction is very often followed by partial restoration of bone, improving support of the adjacent teeth (selective extraction). (See figures 5-11a, b, and c.) This occurs *only* if the adjacent teeth are scaled and root planed at the time of extraction.

5. *Suprabony pockets.* The location of the base of the pocket affects the prognosis of an individual tooth more than the pocket depth. For example, a tooth with minimal pocket depth and extensive recession can present a poorer prognosis than a tooth with a deeper pocket and no recession. Likewise, proximity of pockets to frenal attachments and to the mucogingival junc-

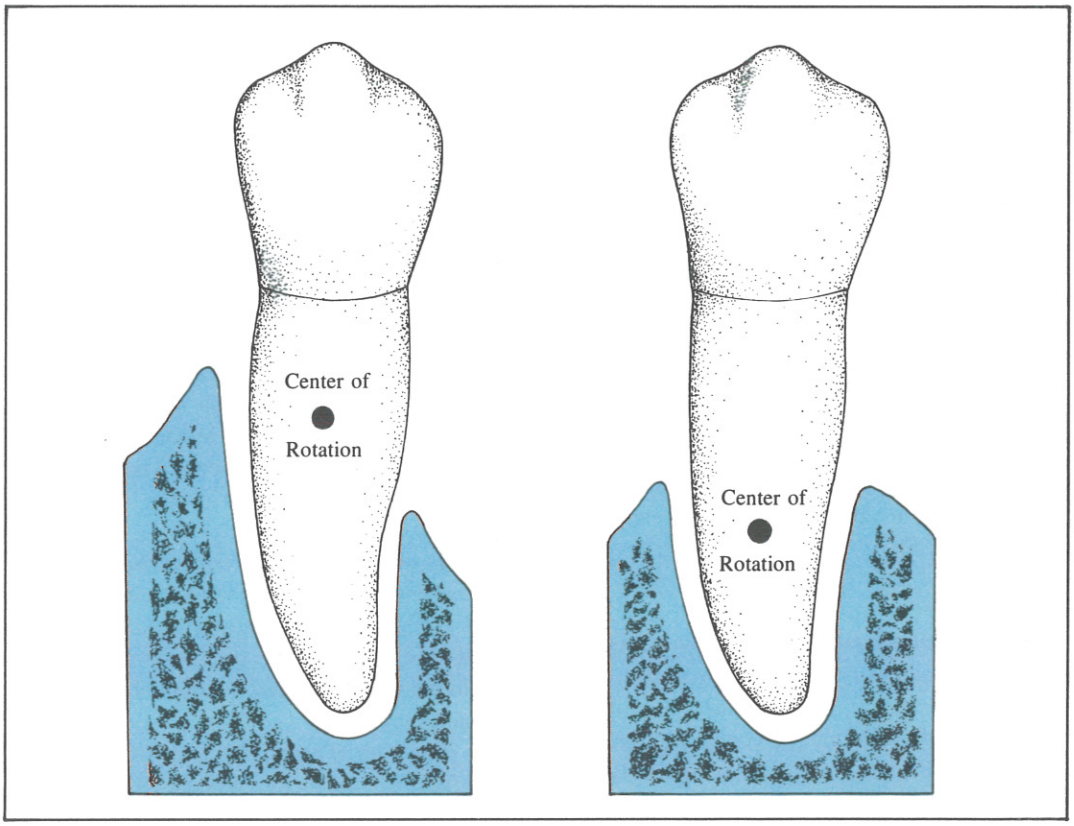


Figure 5-10

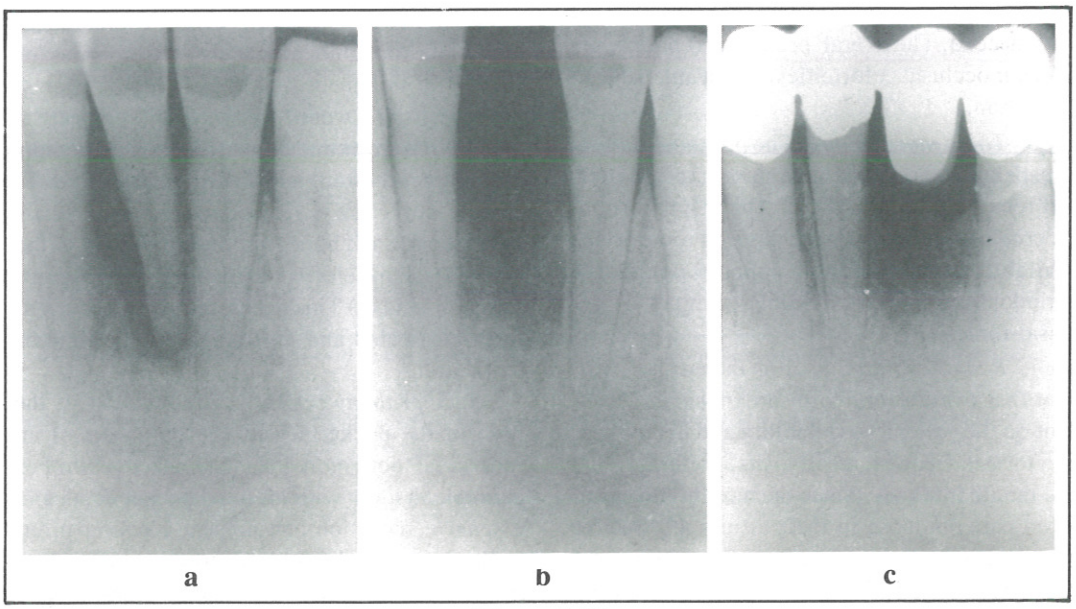


Figure 5-11

tion jeopardizes the prognosis unless corrective procedures are included in the treatment plan. When the periodontal pocket has extended to involve the apex, the prognosis is generally poor. However, significant osseous regeneration is sometimes obtained by combining endodontic with periodontal therapy.

6. *Infrabony pockets.* The likelihood of eliminating infrabony pockets and their associated osseous defects is influenced by the number of remaining bony walls.

7. *Furcation involvement.* Bifurcation or trifurcation involvements must be considered in the prognosis of individual teeth. Furcation involvement does not always indicate a hopeless prognosis. Added support gives multirooted teeth an advantage over single-rooted teeth with comparable bone loss. Several factors influence the prognosis of teeth with diseased furcae:

a. *Extent of furcation involvement.* Early furcation involvement (Class I) affords an excellent prognosis for new attachment.

b. *Access to the furca for surgical management.* A narrow interradicular space offers a poor prognosis for new attachment procedures or root resection because of the close proximity of the adjacent root or roots. It also compromises the plaque control efforts of the patient. Generally, the more divergent the roots, the better is the prognosis; e.g., mandibular second molars have a poorer prognosis than first molar furcations because their roots are shorter and the interradicular space is constricted.

c. *Access to the furca for plaque control.* Generally, mandibular molars with furcation involvement have a better prognosis than maxillary molars with furcation involvement because patients have better access to the mandibular molars. Maxillary bicuspids with furcation involvement are poor candidates for therapy because of root morphology and poor access for plaque control before and after therapy.

8. *Caries, nonvital teeth, and tooth resorption.* In teeth mutilated by extensive caries, the feasibility of adequate restoration and endodontic therapy influences periodontal treatment. Extensive idiopathic root resorption jeopardizes tooth stability and adversely affects the response to periodontal treatment. In treated nonvital teeth, the periodontal prognosis is not significantly affected.

9. *Developmental defects.* Developmental defects, such as the palatogingival groove observed on incisor teeth, present a poor prognosis for successful management. Root concavities observed in some teeth, particularly the maxillary first bicuspid, complicate the prognosis for surgical success as well as for maintenance after surgery.

Treatment Planning

After the diagnosis and prognosis have been established, treatment is planned. The treatment plan is the road map for case management. It includes all procedures required for the establishment and maintenance of oral health.

Periodontal treatment requires long-range planning. The value of periodontal treatment to the patient is measured in years of healthful service of the entire dentition, not by the number of teeth retained at the time of treatment. The treatment plan, therefore, is concerned with the entire dentition rather than with individual teeth. Its principal purpose is to provide a healthy foundation for the future rather than simply to salvage those teeth that were affected in the past. It is directed toward establishing and maintaining the health of the periodontium throughout the mouth rather than toward spectacular efforts to "tighten" loose teeth.

The welfare of the dentition would not be jeopardized by heroic attempts to retain questionable teeth. The clinician is primarily interested in teeth that can be retained with maximum reliability. Such teeth provide the basis for a constructive total treatment plan.

OBJECTIVES OF PERIODONTAL THERAPY

Therapy for periodontal disease has the following objectives:

1. Removal of all etiologic factors.
2. Elimination of all pockets and establishment of minimum sulcus depth.
3. Restoration of physiologic gingival and osseous architecture.
4. Establishment of a functional occlusion by restorative procedures and occlusal adjustment.

5. Maintenance of periodontal health through adequate plaque control by the patient and periodic recall visits to the dentist.

If the clinician can successfully attain these objectives, most cases of periodontal disease can be arrested on a long-term basis.

ORDER OF TREATMENT

The detailed treatment plan must be based on the patient's dental and medical histories, emotional status, clinical and radiographic examinations, and the other factors that have been mentioned. Treatment plans, therefore, have many variations, but in general all consist of four phases: Initial preparation, pocket elimination, restorative treatment, and maintenance.

Initial Preparation

This phase usually includes the following steps:

1. Premedication. Attention is given to the need for premedication for subacute bacterial endocarditis, heart disease, hypertension, and other systemic conditions, as well as preoperative sedation, where indicated. Diabetic patients, or those on special medications, may require special management.

2. Emergency treatment of periodontal abscesses, acute necrotizing ulcerative gingivitis, large carious lesions, etc.

3. Instruction and motivation of the patient in personal plaque control procedures. Success depends primarily on the patient's willingness to participate as a serious co-therapist in his own treatment.

4. Scaling and root planing to remove calculus and enable the patient to begin a program of personal plaque control as early as possible.

5. Extraction of teeth with a hopeless prognosis and extraction of teeth to improve the prognosis of adjacent teeth, accompanied by scaling and root planing of the adjacent teeth.

6. Removal of overhanging restorations, restoration of contacts.

7. Minor tooth movement.

8. Temporary stabilization may be required to facilitate overall treatment or as an aid in determining the prognosis of certain teeth.

9. Preliminary occlusal adjustment and odontoplasty. Obvious gross occlusal abnormalities (plunger cusps, initial prematurities, marginal ridges) should be evaluated early in treatment and corrected if necessary.

10. Evaluation of results. Elimination of etiologic factors may produce sufficient improvement to permit modification of the definitive treatment plan. In this sense, initial preparation may actually be final, adequate therapy. The patient's attitude toward his plaque control responsibility is also evaluated. Additional instruction may be required, even though the patient is making a sincere effort to practice recommended techniques. On the other hand, a patient's failure to cooperate in this critical area should prompt the dentist to modify, limit, or terminate the course of treatment at this point.

Pocket Elimination

This phase of the treatment involves procedures designed to eliminate the pocket either by resection or by relocating the gingival margin, as well as procedures designed to restore missing parts of the periodontium (new attachment procedures).

Restorative Treatment

The restorative phase usually involves definitive occlusal adjustment, operative dentistry, replacement of missing teeth by fixed and/or removable prostheses, and permanent splinting, where indicated.

Maintenance

Patients are carried in the maintenance phase for a lifetime. Most patients who have been treated for moderate-advanced periodontitis will require reevaluation every 3 to 4 months. The length of time between recall appointments is dictated by the level of disease control accomplished by patients at each recall appointment. This phase of therapy is often downgraded by both patient and practitioner, yet it spells the difference between long-term success and failure. Patients become overconfident in their ability to control their own disease. Dentists become more interested in treating new patients than in closely following former patients. A lack of facilities and auxiliary personnel often prevents the establishment of recall systems to monitor the progress, or lack thereof, of periodontal patients. It is understandable, then, that many patients require treatment for advanced periodontal disease who have received extensive treatment 3 to 4 years previously but who have not been effectively evaluated since that time.

Plaque control 6

Effectiveness
Safety
Practicality
Plaque control procedures
  Brushing
  Devices for proximal cleansing
  Aids for cleansing inaccessible areas
  Oral irrigating devices
  Disclosing agents
  The cleansing routine
Teaching plaque control
Plaque control by diet
Plaque control by chemotherapy

As discussed in chapter 2, periodontal disease is a bacterial infection. Many local factors influence its initiation, but inadequate plaque control overshadows all others. In study after study, the worldwide prevalence and severity of periodontal disease are associated with bacterial plaque, calculus, oral debris, and poor oral hygiene. Neglect, then, is the principal cause of periodontal disease—neglect, on the part of the patient, to remove plaque; then, neglect to seek dental treatment; and last, neglect to remove plaque after periodontal therapy, thereby permitting the disease to recur.

Establishment of plaque control is an absolute requisite for the successful treatment and prevention of periodontal disease. This is true for all patients but especially for those whose oral tissues have demonstrated low resistance to microorganisms. Such patients must be meticulous if they are to arrest their disease. Effective plaque control requires that the patient have comprehension and motivation, manual dexterity, and reasonable access to all tooth surfaces. Absence of even one of these requisites will compromise treatment.

The patient becomes involved in his treatment when he decides to accept all responsibility for daily control of plaque on a long-term basis. His involvement is expressed by the action he takes to remove developing bacterial plaque every day.

The question is, "What action is to be taken?"

Almost all of the various plaque control devices have their uses, depending on conditions present in the individual mouth. Generally speaking, any action is desirable if it is effective, safe, and practical. Regardless of the devices and techniques prescribed, the patient must strive to make his actions habitual.

Effectiveness

Effectiveness implies the cleansing of every surface of every tooth. Patients who brush fre-

quently and manage to cleanse the occlusal and facial surfaces, from second bicuspid to second bicuspid, will benefit in those areas, but many other surfaces may remain uncleansed, and their contiguous soft tissues will continue to be exposed to the destructive agents of bacterial plaque.

Many of these patients think their "tooth brushing" is effective and express surprise when their own ineffectiveness is demonstrated. They may simply not be aware of all the surfaces that need cleansing. They may never have thought about, or may never have been told of, the need and methods for cleansing the proximal surfaces or the lingual surfaces of their lower molars, for example. They may not realize that effort, alone, can be wasted. In fact, they may actually be ignorant of what they should accomplish. Their only exposure to instruction in plaque control may have consisted of a pat on the back and a fatherly exhortation to "Brush your teeth three times a day." For that matter, they probably have no understanding of the nature of plaque.

One requirement for effective action is that the patient understand what he is trying to accomplish. Another is that the accomplishment be realized.

Unfortunately, there is no simple way to remove bacterial plaque other than to scrub, or rub, it off with the bristles of a brush; with fibers of floss, tape, yarn, or gauze; or with toothpick or interdental stimulator. As yet, no agent is available that will totally prevent plaque formation or remove bacterial colonies once they have formed.

The effectiveness of any method of plaque control depends also on whether the surfaces to be cleansed are accessible to the cleansing device. The surfaces of a tooth on which the epithelial attachment is at the cementoenamel junction are more readily cleansed than they would be in a deep periodontal pocket. Obviously, there is less tooth surface to be cleansed in the healthy situation, and fewer concavities are exposed to plaque formation. In other words, as pockets form, more concave root surfaces become accessible for bacterial colonization and, inevitably, cleansing difficulty increases.

These problem surfaces continually challenge the patient's efforts to control plaque. The mesial concavity of the maxillary first bicuspid, for example, requires meticulous attention by the postsurgical patient. Furcations and developmental grooves of roots can also frustrate the efforts of the most determined. When evaluating a patient's effectiveness in controlling plaque, one must consider these difficult areas and the possibility that the patient simply cannot satisfactorily control infection in such areas. The patient should be made aware that while complete control of the disease in such areas may be impossible, his efforts may significantly retard the progress of the disease. These considerations further emphasize the desirability of establishing plaque control before disease occurs.

Generally, the effectiveness of plaque control procedures is determined by application of the following criteria:

1. Minimal plaque formation as determined by the Navy Plaque Index (MANMED art. 6-102A(1) (c) (3)).
2. Absence of bleeding on probing and using dental floss.
3. Physiologic color and consistency of gingival tissue.

Safety

The effectiveness of a plaque control proceddure must be weighed against its safety. Any procedure that is inherently destructive should be avoided. Long back-and-forth strokes with a hard-bristled brush, for example, can result in abrasive destruction of gingiva and tooth. Toothpicks, improperly used, can destroy the papillae. Although destructive practices can effectively control plaque, other effective procedures are safe and, therefore, preferred.

Practicality

A safe and effective method of removing plaque is of no value unless it is practical. This depends on the abilities and attitudes of the patient involved. A complicated routine may be practical for a patient with great manual dexterity and zeal but highly impractical for a truly uncoordinated person. If presented immediately in its entirety, the routine may seem impractical for a patient who maintains some skepticism as a result of

past disappointments. If a complicated routine will be necessary to enable this patient to control plaque, the practical approach would be to introduce the routine piecemeal. This gradual involvement during the initial stages of treatment may overcome the patient's skepticism as he begins to see results.

Plaque Control Procedures

BRUSHING

For a thorough discussion of the many different methods of brushing, the reader is referred to current textbooks on periodontics. The Bass method will be considered in this chapter as the preferred method for two reasons:

1. It is safe and effective.
2. More and more practitioners who have had opportunities to make a critical evaluation of all methods of brushing find that best results are obtained with this method.

The Bass tooth brushing technique has also been called "sulcular cleansing" because its intent is to remove bacterial plaque from facial and lingual sulcular spaces. If practiced effectively, the method does result in cleansing the dentogingival junction—the critical area in periodontal disease. Cleansing is accomplished by directing the bristles of a soft brush at the dentogingival junction and moving the brush back and forth in short, almost vibratory strokes (fig. 6-1). The occlusal surfaces are cleansed by back-and-forth scrubbing.

Another integral part of the Bass method is the cleansing of proximal surfaces with floss. Unfortunately, little more than lip service has been given to proximal cleansing. To many people, including many dentists, the term "oral hygiene" means only tooth brushing, with little or no attention directed to proximal cleansing. In an attempt to overcome this almost fixed notion, many practitioners have abandoned the term "oral hygiene" and use the phrases "plaque control," "plaque control procedures," and "plaque control instruction," since proximal cleansing must be accomplished if plaque is to be controlled.

It is gratifying that most patients who are aware of their disease and of its cause, and who are interested in arresting it, recognize the necessity for proximal cleansing when the need is explained by a motivated dentist. Once involved in their own treatment, these patients' efforts can be surprisingly effective.

Manual toothbrushes. Toothbrushes range in size and shape, bristle hardness, arrangement, and length. There are a number of good brushes on the market, and it is a smart clinician who selects a brush that fits the patient rather than fitting all patients to the same brush. Generally, a brush with a straight handle and multitufted, medium-soft, nylon bristles with rounded ends is preferable for use in sulcular cleansing. Patients should be advised that all toothbrushes should be replaced periodically before the bristles become frayed and lose their shape.

Sulcular Cleansing

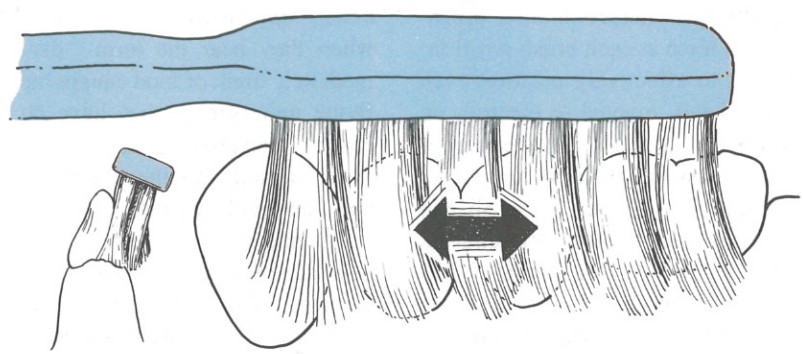


Figure 6-1

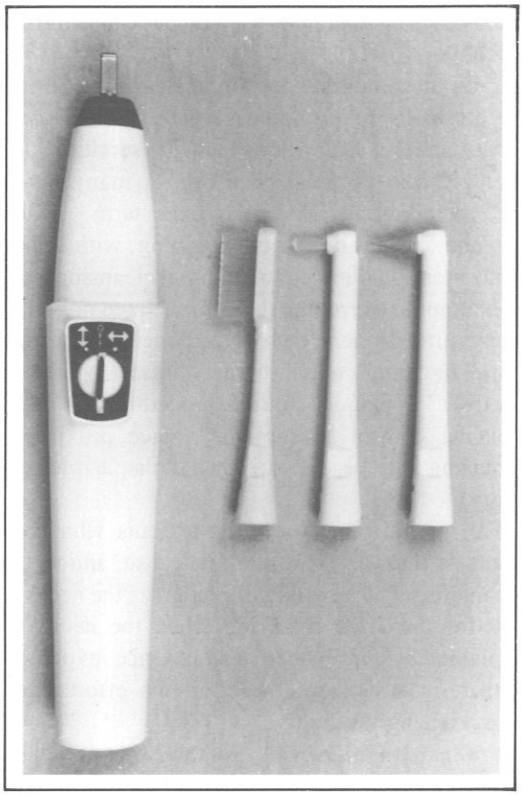


Figure 6-2

Electrically powered toothbrushes. A wide variety of electrically powered brushes are available to the patient. However, a reciprocating-action brush is needed to accomplish sulcular cleansing; a brush with only an arcuating stroke is unsuited for this purpose. The brush shown in figure 6-2 offers both types of motion. It also comes with various attachments that are valuable aids in cleansing inaccessible areas.

The reciprocating action electrically powered brush is positioned exactly as a handbrush. The patient is instructed to permit the bristles to work subgingivally and interproximally, brushing only one or two teeth at each brush position. It is often difficult to effectively position even the smallest brush head, manual or electric, on the palatal and lingual surfaces. The electric reciprocating action brush can be used very effectively in these regions because the brush can easily be inverted and each tooth can be "hooded" with the bristles and brushed individually, as shown in figure 6-3.

Manual vs. power brushing. Numerous studies have been reported concerning the effectiveness of manual and powered brushes. Probably the most important conclusions to be reached are, first, that a motivated, dexterous patient can produce effective results with either kind and, second, that patients with limited dexterity can generally do better with a powered brush. Studies at the Naval Academy, where a reciprocating action powered brush is issued to all midshipmen, have demonstrated its greater effectiveness in comparison with manually operated brushes. The high degree of patient acceptance and the low incidence of mechanical failure are likewise encouraging. When patients demonstrate an ability to use a handbrush effectively or require only minor modification in technique, there is no need to switch. When they have attempted a number of manual techniques over the years and have been unable to master one, consider the electric brush. It is often more rewarding, dentally and psychologically, to introduce an entirely new concept of tooth cleansing, such as the electric brush, than to attempt to break bad habits that have occurred over the years with the manual brush.

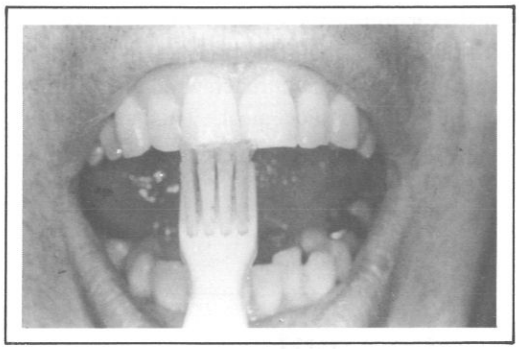


Figure 6-3

DEVICES FOR PROXIMAL CLEANSING

Floss, tape (waxed, unwaxed). Many patients, when they hear the term "dental floss," immediately think of food caught between the teeth. Being uninformed, they have no conception of how to clean proximal surfaces and must be made to understand what they are to accomplish, why, how, and with what devices.

Dental floss and tape, either waxed or unwaxed, are very useful for cleansing proximal surfaces. The difference in effectiveness between unwaxed floss, waxed floss, and tape is an interesting topic of argument, with each proponent convinced of the superiority of one over the

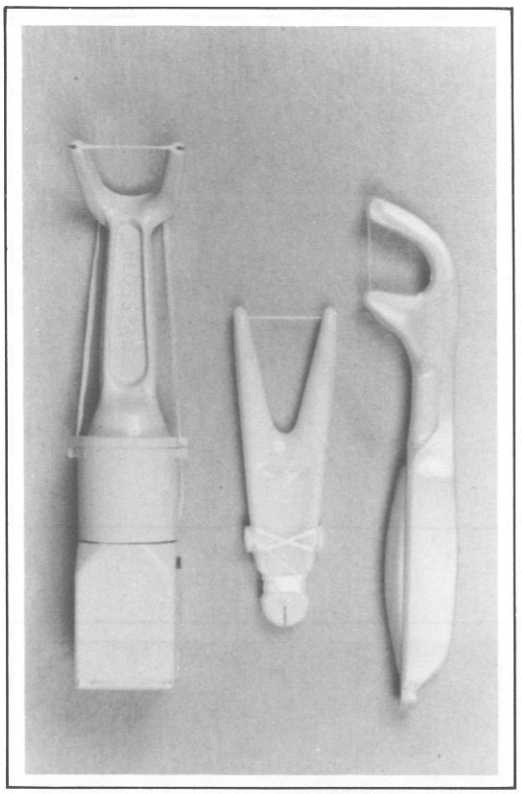


Figure 6-4

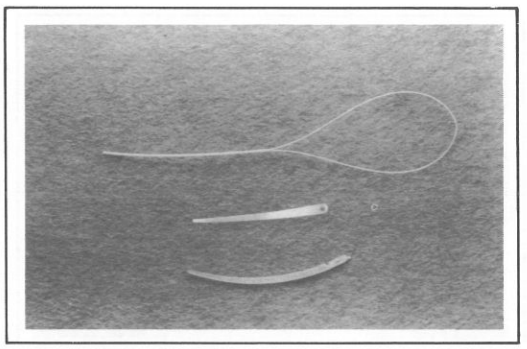


Figure 6-5

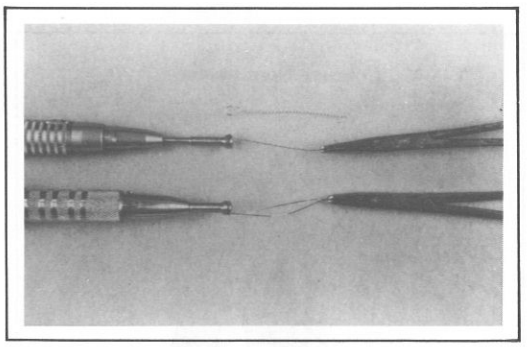


Figure 6-6

other. However, the important factor seems to be what is done with the floss or tape (see NAVMED Publication P-5118 (1973), "Now You are in Charge").

The use of floss presents certain problems which should be explained to the patient. Popping floss between the contact areas of adjacent teeth can lacerate the papilla. However, this damage can be avoided if the user exercises control and slides the floss back and forth between the contact areas while pressing apically. The floss-holding devices shown in figure 6-4 have proved effective for some patients who have difficulty in guiding floss with their fingers. Others find it easier to tie the two free ends together forming a circle, which improves their ability to control the floss. Patients with fixed splinting of teeth are faced with a difficult problem of access to the interproximal region. Floss can be threaded through embrasures by commercially available devices such as those shown in figure 6-5. Suture wire is also used for this purpose and has the advantage that it is easily contoured by the patient to fit the embrasure. Wire threaders are prepared by twisting two strands of 0.010 dead soft wire by means of the dental handpiece (fig. 6-6).

Removing floss in a straight occlusal direction can severely test the retention of restorations when contacts are tight. The problem can be alleviated if the patient either pulls one free end of the floss through the interproximal space or pulls the floss through the contact laterally rather than occlusally. (It should be pointed out that the motivated patient who is meticulous in the use of floss, especially unwaxed floss, will quickly determine the presence of overhanging margins, or sharp edges of restorations or enamel.)

In use, the floss is pressed closely against the tooth and scrubbed up and down, rather than in "shoeshine" fashion, cutting plaque from flat or convex surfaces (fig. 6-7). Problems arise when proximal surfaces are concave and inaccessible to the cleansing fibers (fig. 6-8). Such surfaces require other devices.

Yarn. Two-ply white nylon knitting yarn, used in the same manner as floss, is effective in cleansing smooth and convex tooth surfaces.

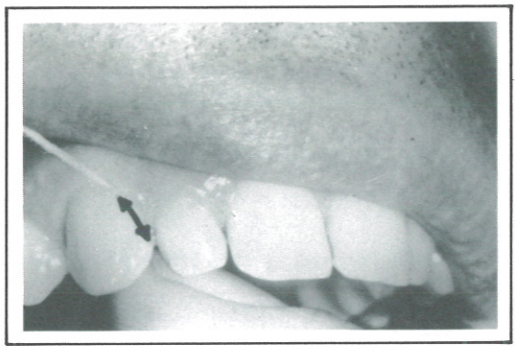


Figure 6-7

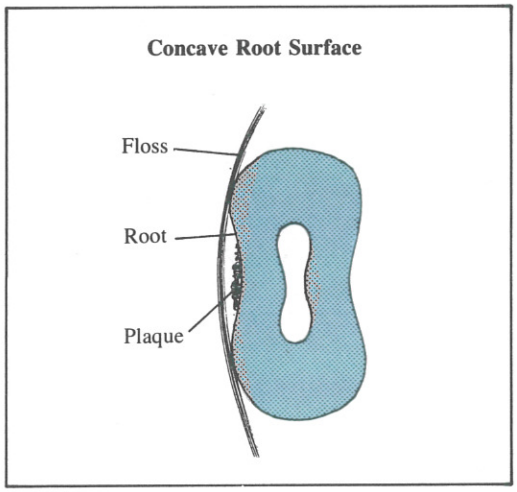


Figure 6-8

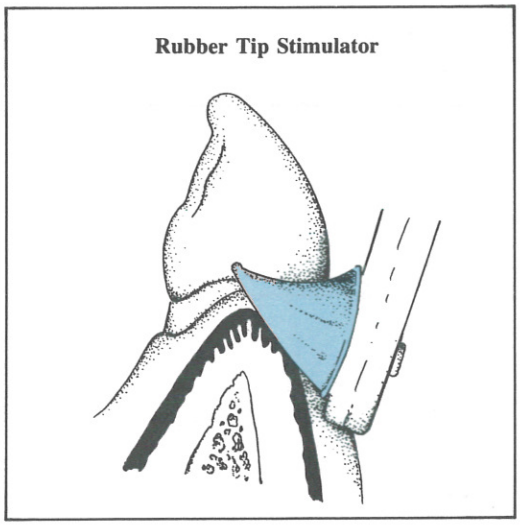


Figure 6-9

Figure 6-10

Gauze. Strips of gauze, in 1-inch or ½-inch widths, are particularly effective on proximal surfaces adjacent to an edentulous space.

Rubber, plastic, and wooden interdental cleansers. Rubber- or plastic-tipped interproximal stimulators can be used to good effect. Many clinicians feel that these devices can be used to promote and maintain healthy gingival contours interproximally, if used as demonstrated in figure 6-9. They are also excellent for cleansing exposed furcations. Rubber-tipped stimulators are available as attachments to the reciprocating action electrically powered toothbrush shown in figure 6-2.

Toothpicks and Stim-U-Dents. These are used successfully by many patients who have been shown how to use them properly. Improperly used, they can damage the papillae. Some patients have difficulty using them in the posterior areas, but these devices are convenient to carry, and their use readily becomes habitual. Those tooth surfaces that are accessible to their action can be made plaque-free.

Although there is no general agreement on the subject, many clinicians believe the effectiveness of all rigid interproximal devices may be in their plaque removal potential rather than through stimulation.

AIDS FOR CLEANSING INACCESSIBLE AREAS

Single-tufted toothbrush. This attachment to the electrically powered brush has been shown to be highly effective, well accepted by the patient, and safe in cleansing such areas as furcations (fig. 6-10), concave surfaces, uneven gingival margins, malpositioned teeth, lingual and palatal

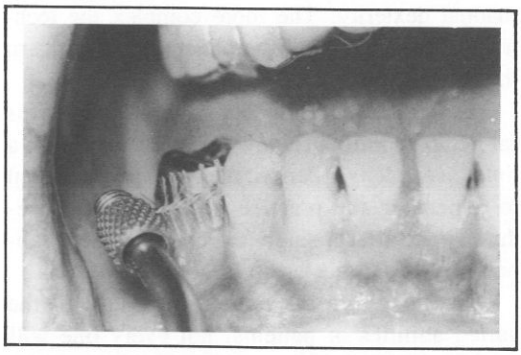


Figure 6-11

surfaces, areas of erosion and abrasion, and around fixed prostheses. Patients are instructed to trace the gingival margins with the bristles directed toward the gingiva and to hold the brush in each interproximal area, permitting the bristles to work interproximally and subgingivally. Inaccessible areas are dealt with individually. A similar monotuft brush can be purchased for manual use.

The Proxabrush (fig. 6-11). This is likewise a useful aid in open embrasures where concavities cannot be adequately cleansed with floss, tape, yarn, or other devices. It includes a metal handle with brush heads of various sizes and shapes.

ORAL IRRIGATING DEVICES

Oral irrigators, whether of the pulsating or steady stream type, are considered as adjuncts to the toothbrush in helping to maintain oral health. They do not remove plaque and, consequently, do not replace the toothbrush *or* interdental cleansing devices. Some reports indicate that irrigating devices may alter plaque, but what this alteration means as far as oral health is concerned has not been demonstrated. The water devices appear to have their greatest advantage in removing loose *debris* from areas which cannot be cleansed with the toothbrush, such as around orthodontic bands, fixed bridges, and periodontal pockets. Studies have shown that transient bacteremias may occur with the use of oral irrigators. The effect of such bacteremias in a healthy patient is unknown, but in a patient with a cardiovascular problem, the possibility of bacterial endocarditis must be considered.

DISCLOSING AGENTS

Disclosing media are important in patient education as well as in accomplishing effective plaque control. It is important that the patient recognize the presence of plaque in his own mouth and equally important that he know when this material has been completely removed. Numerous disclosing agents are available, but an erythrosin solution that can be compounded at your local pharmacy is recommended. Its formula is as follows:

Erythrosin ..... 5.6 Gm
Aqua D .....400 ml
Alcohol (95%) .....40 ml
Raspberry flavor ....35 drops

This will make about 14 ounces.

Generally, solutions stain more intensely and last longer than tablets. Patient acceptance is also better.

Some patients will object that their lips, tongue, and mucosa remain stained for some time after use of the erythrosin solution. If this problem discourages use of a disclosing dye, it is recommended that the patient be encouraged to use a disclosing solution, such as Bismark brown, which is not as objectionable to most patients. Another alternative is to use a fluorescent solution, FD and C yellow No. 8. By means of a special dichroic filter placed over the bathroom mirror light, the plaque will fluoresce, and patients can view plaque with no aftereffects. A special light can also be purchased for patient use in viewing the fluorescing plaque.

THE CLEANSING ROUTINE

Plaque removal is accomplished in either a patterned or a nonpatterned manner.

Nonpatterned. In the nonpatterned manner, the patient is told, in effect, to "get the red off." The patient is directed to rinse with a disclosing solution and then rinse with water. Then, with good lighting and a face mirror, he uses his plaque-control devices to remove all of the stained material from his teeth and tongue. A mirror light and a mouth mirror are helpful adjuncts for viewing the stained plaque. When he has finished, he can restain to check his effectiveness and cleanse again if necessary.

Patterned. The patterned manner stresses repetition of the same cleansing routine daily with the hope that the action will become habitual. For

example, the patient is directed to use his proximal surface plaque-removing device first, starting at the last maxillary right molar and going around the arch to the last maxillary left molar. He then proceeds from the last mandibular left molar around to the last mandibular right molar. Next, he scrubs the occlusal surface with a brush, using 10 back-and-forth strokes per quadrant in the same order: upper right, upper left, lower left, lower right. He next uses the soft brush, applying 10 short, vibratory strokes in each area, to cleanse all facial and lingual surfaces in the same sequence: upper right, upper left, lower left, lower right.

The patient is advised to brush his tongue in order to reduce the colonies of microorganisms that can contribute to the re-formation of plaque on the tooth surfaces.

Unfortunately, few patients are able to control plaque completely on their first attempt. They must be seen regularly early in treatment for evaluation of their efforts and for additional instruction, guidance, and encouragement. Some dentists have successfully based their entire practices on a foundation of plaque control. They arrange for their patients to participate in a program in which they are seen daily for 5 consecutive days. It is unfortunate that some patients misconstrue the significance of the 5-day appointment schedule, and wording of a certificate for achievement of effective plaque control, to mean they will no longer require instruction or assistance in plaque control in the future. Patients' efforts at plaque control must be constantly evaluated from the standpoint of both effectiveness and safety, and they should be so informed.

Teaching Plaque Control

There are many approaches to teaching patients how to perform effective plaque control. No single technique has been devised that will satisfy the needs of every patient or that can be taught by every clinician. There are certain fundamental principles, however, that can be applied to virtually every patient. They are:

1. *Keep instructions simple.* Remember, practicing plaque control is really an exercise in manual dexterity. The more complex the techniques, the more skill the patient needs to learn them.

2. *Do not teach too much at one time.* It is far better to introduce new techniques a few at a time, over a longer period, than to expect a patient to remember and practice a long list of procedures that, after a single exposure to them, appear very complicated to him.

3. *Encourage the patient.* Because of the varied abilities of patients, not every one will perform adequate plaque control at first. With further assistance and continued encouragement, almost all patients can be motivated to practice better oral hygiene. Do not, however, excuse lack of effort. One must differentiate between lack of willingness to improve and lack of knowledge and skill.

4. *Continue observation and supervision.* No matter how well a patient practices plaque control after the initial instructional program, repeated professional evaluation is required to help him maintain a high level of performance. The interval between these supervisory evaluations will vary from patient to patient.

5. *Be flexible.* Although you may teach a specific technique to all patients, remember that not all patients have the same problems. Crowded or widely spaced teeth, length of crowns, presence of fixed prosthetic appliances, and physical disabilities are only some of the variables encountered. Be prepared to alter techniques, and be knowledgeable about products available for use as adjuncts in controlling plaque.

Plaque Control by Diet

It is an accepted fact that the ingestion of sucrose increases plaque formation. Bacteria form dextrans from carbohydrates, particularly sucrose, and plaque grows in abundance. It is often more difficult to motivate patients to reduce their carbohydrate intake than to induce them to practice daily effective plaque control. They should be warned, however, of the consequences of consuming sugar and sugar-sweetened foods and be encouraged to use substitute foods (diet drinks, sugarless gum, unsweetened juices, etc.).

Plaque Control by Chemotherapy

Worldwide research into the control of plaque by drugs is being performed, with stress on disinfectants, antibiotics, and enzymes. The development of a harmless agent that would prevent plaque formation and remove formed plaque is an attractive concept and would have tremendous impact on dental disease. A number of drugs are presently under study, but considerable research is needed before these agents are available for general use.

Recent studies have demonstrated that a commercial mouthwash* is capable of significantly reducing plaque formation and gingivitis. This mouthwash is swished in the mouth for 30 to 60 seconds three times daily, after breakfast, lunch, and before retiring. The major complaint from patients is the taste. Some also complain of a burning sensation when it is held in the mouth. The value of this mouthwash is that it is commercially available and can be used by patients who cannot effectively cleanse their teeth manually. It is advocated only as an adjunct to regular plaque control procedures, never as a replacement for tooth cleansing procedures.

In considering chemotherapeutic management of plaque infections, it should be remembered that this is still in the future. Today, the only proven plaque control method is physical removal of accumulated materials by tooth-cleansing devices. Therefore, successful control of the world's most common diseases, caries and periodontal disease, still depends upon effective action by the patient who has been motivated, educated, and assisted by his dentist!

*Contains boric acid, menthol, thymol, eucalyptol, methyl salicylate, alcohol 25%.

Patient education and motivation

7

The problem
The technique
  Ignorance
  Awareness
  Interest
  Involvement
  Action
  Habit
Reinforcement
Educational and motivational environment
Use of educational materials

The most important and difficult phase of periodontal treatment is the effective motivation of the patient, so that he endeavors daily to eradicate the cause of his disease. In all other phases of treatment, the dentist has much greater control over what is accomplished. He can scale teeth, plane roots, perform surgery, splint teeth, adjust occlusion, and accomplish any number of other intricate periodontal procedures. In the absence of daily plaque control, however, all these phases of treatment, regardless of how well performed, will be merely palliative and will contribute little to long-term periodontal health.

The Problem

Until such time as measures are available that effectively prevent destructive microbial activity, independent of action by the patient, it will be necessary for the patient to act. The problem is to get the patient to act effectively.

It would be difficult to find a patient who does not know that he should brush his teeth. Indeed, many servicemen have been instructed in brushing, first in elementary school, then by their family dentist, again when they were in basic training, and perhaps later in their service by dental officers. Many patients brush their teeth every day but still have periodontal disease. Their teeth are over-brushed and under-cleansed.

It is easy to get people to brush their teeth. On the other hand, it is a challenge to get them to cleanse their teeth every day. In this sense, the term "to cleanse" means "to remove colonizing bacteria from every surface of every tooth every day."

Somehow, the patient must be motivated to such a degree that he makes a conscious decision

to control plaque on a daily basis. If he wants to establish and maintain oral health, the patient must make a positive, long-lasting decision. He must be concerned with health, not simply for the relatively short time he is undergoing active treatment, but for the rest of his life. The ideal goal would be to have the patient control bacterial plaque so well that he would never require the assistance of a dentist regarding his periodontal health. Although this goal may be unattainable, every patient should at least be considered as a candidate for such an accomplishment.

It is impossible to evaluate any patient's capacity for motivation at the first appointment. Some patients, whose literally filthy mouths seem to cry out for demolition, prove to be able and willing to control plaque effectively. Many of these apparently negligent patients have never really understood why or how they should cleanse their teeth, nor have they ever realized the potential benefits of their own efforts.

On the other hand, some patients will never be willing to devote a portion of their day to effective plaque control, despite the dental officer's best efforts. It is well to recognize that there are such patients and that, for them, it will be impossible to establish and maintain periodontal health. Surgical correction of existing tissue deformities, for example, would subject these patients to useless surgery and uncomfortable periods of healing at the expense of time that could more profitably be spent with motivated patients.

Unfortunately, effective motivation of a patient is neither a particularly easy task nor a quick one. Adequate physical facilities, trained personnel, and enthusiastic cooperation by those in command are essential for effective motivational efforts directed toward groups. Even when a group program is fully operational, however, it is still the responsibility of every dental officer to devote time to motivating each of his own patients in plaque control.

To effectively motivate his patients, the dental officer must himself be convinced that daily removal of the bacterial colonies from all tooth surfaces is worthwhile. If he is not, it is questionable whether his approach to the problem of motivation will be other than casual, and a casual approach to motivation is always a waste of time. On the other hand, if the dental officer approaches the problem with the firm intention of impressing his patient to such an extent that he will establish a habit of daily plaque removal, then the time will be well spent.

One definition of the word "habit" in Webster's New World Dictionary is "a thing done often and, hence, usually, done easily." Another is, "an addiction: as, the alcohol habit." Whether many patients will find plaque control habit-forming is problematic. It is more likely that, as a result of efforts by the dental officer, many will begin to control plaque daily, will do it often and, it is hoped, easily.

Unlike many habits that are acquired unconsciously, the habit of daily plaque control is the result of a deliberate, conscious decision by the patient. The dental officer's problem is to ensure that his patient makes this decision.

The Technique

The habit of plaque control can probably be best achieved by a technique successfully used in allied fields, called the decision continuum technique.* In this technique, decision-making, with habit as the ultimate product, is a six-step process: (1) ignorance, (2) awareness, (3) interest, (4) involvement, (5) action, and (6) habit.

The first step is a state of *ignorance*, a starting point for decisive action. The second step requires awakening the individual to *awareness* of his problem and of the need for a solution to that problem. The next step is to gain his *interest* in the problem and to maintain his interest in possible solutions on a continuing basis. Awareness and interest lead to the most important, difficult, and overlooked step of the process, which is the *involvement* of the individual in the solution to his problem. In other words, he must be persuaded to participate in the therapeutic resolution of any existing or future periodontic problem.

Once the patient becomes involved in his own problem, *action* can easily be stimulated by encouraging him to act on the solutions to his problem and to persevere until he has established a *habit* pattern in carrying out the therapeutic

*Cassidy, R. J. Psychological factors in preventive dentistry. Ala. J. Med. Sci. 5:358–369, 1968.

procedures that have been proposed to solve that problem.

IGNORANCE

Most patients are totally ignorant of the etiology of dental disease and are unaware that, with proper care, the human dentition can last a lifetime. Patient education is an important step in the process of motivation, and it is closely related to the second step of the technique, creative awareness.

AWARENESS

The patient with advanced periodontal disease is usually aware that his mouth is not totally healthy. He may have accepted his periodontal disease as inevitable, as a result of either age or heredity, or perhaps because he has been "treated" unsuccessfully in the past. This kind of awareness is inadequate as a help in decision-making directed toward health. The patient must be made aware of the specifics of his disease. To detect the disease signs, a hand mirror is essential. He must actually see the inflammation, bleeding, exudate, mobility, and loss of gingival attachment (pockets and recession) in his own mouth, and he must understand that these signs are related to bacterial plaque. Ideally, he will see the microorganisms in his plaque through a phase-contrast microscope.

The phase-contrast microscope is a valuable tool in motivation for plaque control. Most patients understand the cause-and-effect relationship of bacteria and disease but have never associated microorganisms with dental disease. A patient can make the association if material is removed from his own teeth, placed on a slide, and then viewed through the microscope. Seeing his own living, moving microorganisms, the patient is often startled into an awareness of the unhealthy state of his mouth.

The patient with early or moderate periodontal disease may be unaware of his condition. Although he presents a greater challenge than the patient with advanced destruction, he can usually be made aware by use of the same techniques.

The patient with no clinically demonstrable sign of periodontal disease obviously cannot be made aware of disease in his own mouth, and therefore a greater problem is presented. He can, of course, be made aware of what periodontal disease is by means of drawings, photographs, pamphlets, etc. He can be informed of the possibility that he will develop the disease. He can also be shown plaque from his own mouth, but it is difficult to convert future possibilities and nonmotile plaque into very potent motivators. Perhaps experiences of his relatives or friends can be used to increase his awareness. At any rate, the interested and ingenious dental officer should be able to establish some degree of awareness in most patients.

INTEREST

Interest in the problem and in its resolution is another matter. Patients who present with complaints arising from their disease demonstrate some degree of interest. Some may indicate great concern and a willingness to do anything to save their teeth. Others may only seek relief from an emergency condition. This latter type of patient may not be interested in long-range health, but once he is made aware of the reality and extent of his disease, he may become more interested in his problem.

A more difficult patient is one who apparently just doesn't care. He is not in pain; he eats well; his father and mother have a full set of dentures and get along fine; "besides, he's always had soft teeth, and gum disease runs in the family and, anyhow, he's had treatment forced on him before" and his attitude is, frankly, "Leave me alone."

Occasionally, a patient is found whose lack of interest is real, established, and unalterable. For this individual, extended treatment is contraindicated. However, many of these apparently uninterested patients can be stimulated by a conscientious dental officer who is interested in and concerned about the patient's well-being. Whether the appeal is based on the patient's desire for health, social standing, or employment opportunities, on his sex life, his pride of accomplishment, or any other source of interest, depends on that individual patient's attitudes and those of the dental officer.

INVOLVEMENT

A patient who is aware that he has periodontal disease and is interested in establishing oral health is a good candidate for success. But awareness and interest, alone, are not enough to ensure success. Many patients with advanced

periodontal disease have sought and received dental treatment throughout their lives, but for some reason, they have never really become actively involved in their therapy.

Patient involvement in therapy implies daily removal of plaque from every surface of every tooth. In most instances, it is an action that only he can provide. Yet, how disheartening it is to one who has "brushed" his teeth three-times-a-day throughout the years to find that he has developed advanced periodontal disease with destruction particularly evident interproximally, where plaque has grown undisturbed for a long time. Early involvement in plaque control could have channeled his efforts productively and prevented such destruction.

ACTION

If the patient is truly aware, interested, and involved in therapy, then action is automatically ensured. Whether this action becomes habitual will depend, to a great extent, on the dental officer's ability to instill in his patient the desire to establish long-lasting health. Unfortunately, there is a natural tendency for this ability to be diluted by monotony. The information presented to the patient concerning his disease—what it is, and what can be done about it by the dentist and by the patient—is really quite simple. The danger lies in the presentation's becoming stale. Similarly, enthusiasm for success *must* be communicated to the patient, and it will remain fresh only if the dentist continually renews his own excitement about how much the patient can accomplish by himself. The dentist must develop a patient's confidence, so that the patient is aware that he has the power to control his own disease. It is critical for both people to understand that visible evidence of disease control will be reflected within a week by decreased bleeding, changes in gingival color and form, tightening of teeth, better taste, etc. Plaque control is not academic!

HABIT

The establishment of a habit pattern for daily removal of bacterial plaque is the end result of the successful application of the decision continuum technique. To achieve this goal requires salesmanship of the highest order. Once achieved, reinforcement is necessary to maintain this habit.

Reinforcement

Most information and habits that are not continually reinforced are usually quickly forgotten, and plaque control is no exception. The number of appointments necessary to initiate interest and awareness within a patient is highly variable. It is unlikely that a single experience is beneficial. Programs in which patients are seen on 5 consecutive days appear successful initially, but the long-term results will still depend on reinforcement throughout life. If plaque control concepts are to be retained and habit patterns established, reinforcement should be accomplished at every routine dental appointment or examination period during a military career.

Educational and Motivational Environment

The decision continuum technique can be initiated in a number of environments. Some practitioners believe that patients are more easily motivated on a one-to-one basis and feel quite confident in using their dental chair to educate and motivate patients. Others believe that patients are more receptive in a specially designed plaque control room. Certainly, teaching becomes much easier in a room which permits the free utilization of a variety of audiovisual aids and a sink and a mirror for the patient to practice the various techniques.

There are some studies which indicate that small groups (6 to 8 patients) can be taught plaque control more effectively than single individuals, provided that those within the group are about the same age and share similar dental problems. Similar grouping of individuals to achieve a common goal is widely used by organizations such as Alcoholics Anonymous, Weight Watchers clubs, and athletic achievement groups. One such plaque control center which employs group therapy is located at the Naval Academy. Here, midshipmen in groups of six are made *aware* of their individual problems with the aid of several audiovisual devices. The midshipman becomes *interested* because his friends share the same

problem. The young subjects become *involved* in the treatment of their own condition. They are checked periodically to ensure *action*. *Reinforcement* of plaque control concepts is accomplished at every routine dental appointment by all dental personnel.

Use of Educational Materials

There are many types of written materials available as handouts for patient education and motivation. The patient should be advised to take the time to read the booklets for comprehension. As casual handouts, they may or may not prove beneficial; but as interesting items for study, they should provide valuable reinforcement to the information already provided in the dental office.

1. The Navy has prepared three pamphlets which serve as excellent handouts: "Now You Are In Charge," NAVMED P-5118 (1973); "The Navy Periodontal Screening Examination," NAVMED P-5111 (1972); and "Do You Hesitate to Smile?" NAVMED P-5117 (1973).

2. Another useful handout is a 32-page, soft-cover booklet entitled "For 9 Out of 10 Adults Only," priced at 25 cents a copy and available from:

Semantodontics, Inc.
4513 North 32nd Street
Phoenix, Arizona 85018

This booklet is the result of a project in programmed instruction by the University of California School of Dentistry in which many approaches to the problem of patient education were explored. The format combines the best features of many other learning aids and is comprehensive and accurate.

3. Two excellent, short informative pamphlets, prepared by the Information Office, National Institute of Dental Research, National Institutes of Health, Bethesda, Maryland 20014, are: "Research Explores — Combat Zone in Dental Disease" and "Research Explores Pyorrhea and Other Gum Diseases — Periodontal Disease." Single copies are available at a cost of 20 to 30 cents each.

Scaling and root planing

8

Introduction
- Definitions
- Cementum removal
- Presurgical scaling

Armamentarium
- Anesthesia
- Technique for hand instrumentation
- Basic strokes
- Ultrasonic scaler

Instrument sharpening
- Sharpening stones
- Felt wheel
- Principles of sharpening
- Test for sharpness

Introduction

DEFINITIONS

Scaling: the procedure by which mineralized deposits are removed from the tooth surface.

Root planing: a more definitive form of scaling to smooth roughened root surfaces.

It is often difficult to determine where scaling stops and root planing begins, and, frequently, the two procedures cannot be dissociated.

CEMENTUM REMOVAL

Opinions vary as to the amount of cementum to be removed in root planing. One school of thought holds that all exposed cementum and "softened" dentin should be removed. Another holds that root planing is unnecessary and that overinstrumentation leads to excessive root sensitivity. All clinicians agree, however, that calculus should be removed, and it is probable that, in most instances, complete calculus removal with a sharp curet will result in the removal of any porous cementum. If this is so, the removal of cementum in its entirety is of academic interest only, provided the root surface is smooth, free from calculus, and can be adequately cleansed by the patient.

PRESURGICAL SCALING

There has been much discussion in recent years about the merits of presurgical scaling. Some therapists have stated that it is unnecessary and causes added inconvenience and discomfort to the patient. The vast majority of clinicians, however, believe that presurgical scaling is an important step in periodontal therapy. Preoperative scaling and root planing reduce inflammation and eliminate the problem of excessive bleeding at the surgical site. Likewise, the tissue will be

firmer and easier to manipulate during periodontal surgery. Clinical experience demonstrates that, after scaling, root planing, and plaque control, some areas will no longer require surgical intervention. Probably the most important reason for presurgical scaling is to create a clean, smooth root surface that will permit the patient to accomplish effective plaque control. If the patient does not perform this all-important phase of treatment adequately, then it is futile, from the standpoint of both the therapist and the patient, to proceed to the surgical phase of treatment.

Armamentarium

The successful practice of periodontics is centered around the skillful use of instruments during scaling and root planing. These instruments generally fall into the broad categories of chisels, hoes, sickles, files, curets, and ultrasonic scaling devices. Calculus deposits can be removed by any one of the six classes of instruments. Curets, however, are the instruments of choice for root planing. It has been demonstrated that the ultrasonic scaler will not plane roots. Hence, this procedure must be accomplished by hand instrumentation, using sharp curets with medium-length strokes executed in a smooth, rhythmic, and continuous manner. There should be "stepping" or overlap of areas around the tooth in order to cover the entire root surface. Care must be taken to avoid scratching or gouging the root.

ANESTHESIA

For most patients, scaling and root planing can be accomplished without anesthesia. In some cases it will be necessary to introduce 5 percent topical lidocaine ointment into the sulcus or pocket a *minimum* of 5 minutes before instrumentation as well as during scaling and root planing. The topical anesthetic dyclonine hydrochloride is also very effective. When topical anesthesia is inadequate, as in the case of patients with extreme tactile sensitivity, block or infiltration anesthesia is recommended.

TECHNIQUE FOR HAND INSTRUMENTATION

Proficiency in instrumentation is obtained by adhering to the following basic principles:

1. *Work comfortably.* Make the patient comfortable, but mainly make yourself comfortable.

2. *Follow an orderly sequence of instrumentation* to avoid omitting a particular tooth surface.

3. *Operate with maximum visibility.* Wherever possible, it is best to have direct vision of the operated area. Also, have a good light source. The high intensity, low heat fiberoptics are very helpful in obtaining visibility. Fiberoptics can also be used for transillumination and may show small deposits that might otherwise be overlooked.

4. *Obtain maximum accessibility* by proper use of mirror, hands, etc.

5. *Maintain complete control of the instruments.* Stability is essential for effective, controlled action of the instrument.

6. *Maintain a clear field* by using gauze and cotton rolls, and by frequent flushing with water, or sometimes air. Flushing is helpful in ensuring that no calculus remains in the gingival sulcus or pocket.

7. *Be certain all instruments are sharp.* A dull instrument will merely slide over the thinner pieces of calculus, giving the impression that all calculus has been removed. Instruments must be sharp to be effective. Sharpen them after each scaling and, sometimes, during scaling.

8. *Be gentle and careful.* Don't confuse roughness with thoroughness.

9. *Know the function of each instrument.* This makes the job quicker and easier.

10. *Use as few instruments as possible.* You become more efficient when using a few instruments. This also reduces the time of scaling and makes the operation easier.

11. *Know the relation of the instrument to the tooth and periodontal structures before activating it.* Put the instrument into place slowly and deliberately. This prevents undue injury to the tissues.

BASIC STROKES

There are three basic strokes for scaling and root planing: the exploratory stroke, the working stroke, and the circumferential stroke.

1. *Exploratory stroke.* This is used to determine the topography of subgingival deposits. The instrument blade is passed along the root surface to the depth of the pocket (fig. 8-1a). If

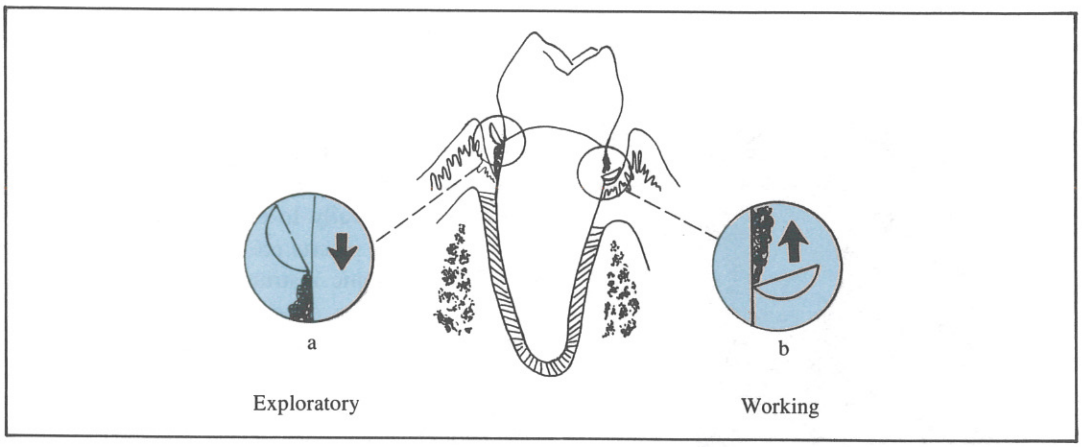


Figure 8-1

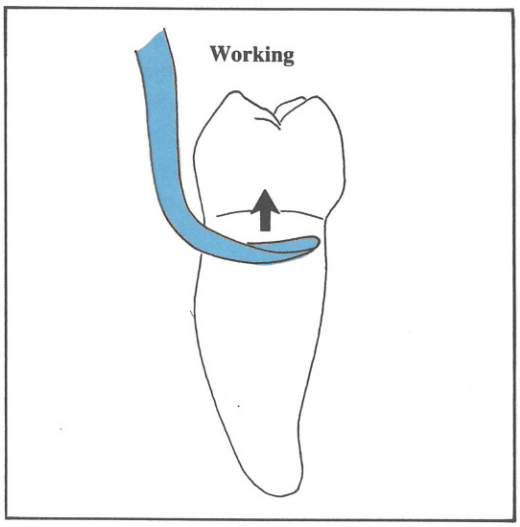


Figure 8-2

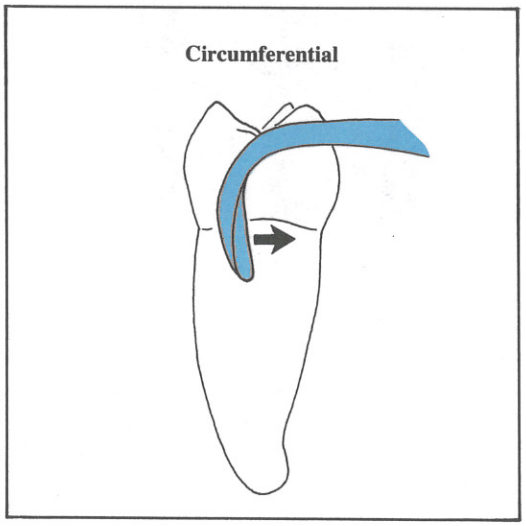


Figure 8-3

any apparent obstruction is encountered during exploration, the blade should be moved laterally from the root surface and, if possible, gently extended apically. This movement will aid in distinguishing between a ledge of calculus and the base of the pocket.

2. *Working stroke*. Once calculus is located, it is removed by engaging the root surface and calculus at an 85° angle (fig. 8-1b) and then moving the instrument coronally along the root. This stroke is followed by a smoothing, shaving action done with absolute control (fig. 8-2). The shaving action is continued until the root surface is completely smooth.

3. *Circumferential stroke*. Certain problem areas, such as line angles of molar roots, depressions, and root surface irregularities, making planing in an apical-coronal direction difficult and, sometimes, impossible. In such areas the circumferential stroke is a valuable adjunct to the basic working stroke (fig. 8-3).

ULTRASONIC SCALER

The ultrasonic scaler provides a fast and easy means of debridement with a high degree of patient comfort. The ultrasonic unit converts ordinary alternating current (110-120 volts, 50/60 Hz (Hertz)) into 25,000 Hz. This current is con-

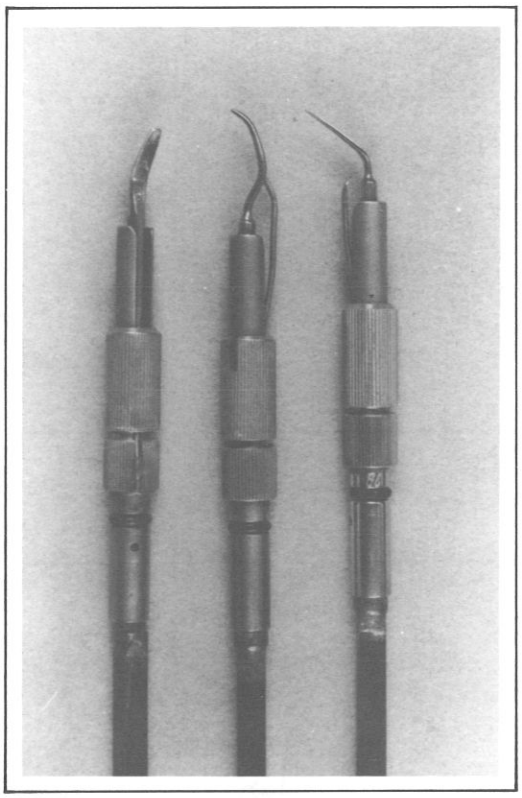


Figure 8-4

verted to 25,000 mechanical strokes per second at the tip of the instrument over a distance of 1/1000 inch. The combined effects of cavitation by the water and vibration by the instrument provide the force necessary to dislodge accretions. No damage to soft or hard tissue will occur if light pressure and adequate water spray are used. Patients experience little or no discomfort as long as adequate water is used and the tip is kept in motion.

The inserts commonly used for ultrasonic scaling are the P-3, P-10, and EWPP (fig. 8-4). The P-3 is excellent for supragingival calculus and can be used to remove subgingival calculus in the interproximal regions. The P-10 can be used anywhere in the mouth and is effective on subgingival calculus. It is used with either a working or a circumferential stroke. The EWPP is best suited for the debridement and curettage of deep pockets. It can gain access to areas that other instruments cannot traverse because of its similarity in design to a periodontal probe. The tip or corner of this insert is potentially dangerous, as are all sharp edges or corners on ultrasonic inserts. Even a dull tip will gouge teeth and restorations if used improperly. It is possible to cut a cavity preparation in a tooth with the tip of an ultrasonic insert, and for this reason it should be used only by experienced personnel, and then with caution. Inserts should be inspected periodically to ensure that the corners and tips of the inserts are dull.

The ultrasonic instrument is highly recommended for gross debridement, particularly in case of necrotizing ulcerative gingivitis or acute gingivitis, where it is the instrument of choice. The instrument removes debris rapidly, with little patient discomfort, while the water spray clears the operative site.

Some clinicians advocate the use of the ultrasonic instrument for debridement during periodontal surgery. A washed field with improved visualization is helpful during surgery; however, the water source from the dental unit often contains microorganisms which could be introduced into the surgical site. If the ultrasonic instrument is to be used during any surgical procedures, it should be used as a self-contained, sterilizable unit with its own source of sterile distilled water maintained in a sterile pressure tank. Even then, it is necessary to routinely check the tank and hoses leading to the insert for possible contamination.

The ultrasonic scaler will remove deep calculus, but gaining access to the calculus is difficult, and there is an absence of tactile sensitivity. The deeper the pocket, the greater is the chance of blockage of water spray to the tip, and, consequently, the greater the possibility of patient discomfort. Ultrasonic instrumentation will remove the pocket lining, if subgingival curettage is the objective, and healing of the wound is as rapid as after hand instrumentation. The ultrasonic scaler will not plane roots, and reports indicate that bacteremias occur as frequently after ultrasonic scaling as after hand scaling.

Dental technicians adapt quickly to ultrasonic instrumentation and can become proficient in its use with proper instruction. Inexperienced assistants, however, should never be permitted to use this potentially destructive instrument. The instrument has been a tremendous asset in the Navy's Preventive Dentistry Program and has saved time in preparing patients for stannous fluoride treatments. The ultrasonic in-

strument is not effective in stain removal; time should not be wasted in removing stains that could be removed quickly and more effectively with a rubber cup and pumice.

It should be emphasized that the ultrasonic instrument is an excellent adjunct in periodontal therapy—but only an adjunct. One simply cannot perform deep scaling or root planing effectively with the ultrasonic tip.

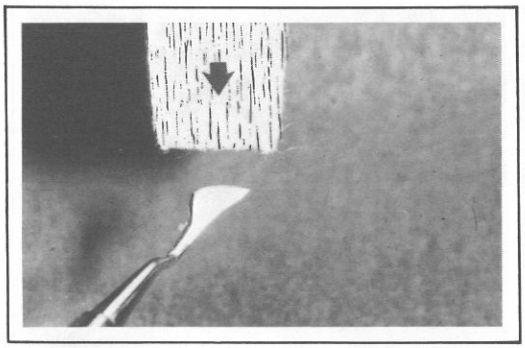


Figure 8-5

Instrument Sharpening

G. V. Black stated that:

*Nothing in the technical procedures of dental practice is more important than the care of the cutting edges of instruments. No man has ever yet become a good and efficient dentist until after he has learned to keep his cutting instruments sharp. . . . The student who cannot, or will not, learn this should abandon the study of dentistry.**

Instruments are sharpened by grinding or polishing the surfaces that form the cutting edge of the blade until the edge is fine and smooth. Cutting edges may be formed by the junction of two plane surfaces, two curved surfaces, or a curved and a plane surface, but regardless of the type of edge involved, the basic principles of sharpening can be applied.

SHARPENING STONES

The first requisite for sharpening a cutting edge properly is to select the correct sharpening stone. These stones are made in various grits (or textures) and in various designs to meet particular needs.

Ruby stone and Arkansas stone are the two types most commonly used for sharpening dental instruments. Ruby stone is fairly coarse, cuts rapidly, and is used chiefly for preliminary sharpening when an instrument is very dull. Arkansas stone is finer in texture than Ruby stone and can be used alone to attain a satisfactory edge.

Sharpening stones may be grouped by design and method of use, as mounted or unmounted. Some mounted stones are inserted in a dental handpiece. Others are used with a specially constructed motor for the laboratory bench and with devices that maintain the desired angles. Unmounted stones may be (1) rectangular stones of different sizes, with surfaces that are either flat or have four different-sized grooves running lengthwise, or (2) cylindrical or specially shaped stones that are activated by hand during the sharpening process.

FELT WHEEL

A medium hard felt wheel, 3 to 3½ inches in diameter and ¾ inch thick, is especially efficient for sharpening knives. Chrome rouge is applied to the wheel as needed (fig. 8-5).

PRINCIPLES OF SHARPENING

To ensure correct sharpening of curets, the following principles should be observed.

1. *Sharpen curets on the sides (lateral edge) and not the face.* Reducing the side narrows the instrument and maintains the thickness between the face and the back, thus ensuring strength (fig. 8-6).

2. *Do not overheat this instrument.* The temper of steel is lost when overheated, and the instrument will not retain sharpness.

3. *Reduce the entire arc of the lateral edge.* If only a segment of the arc is ground, it is difficult, if not impossible, to keep the cutting edge against the root surface (fig. 8-7).

4. *If you are using a stationary stone, pull the side of the instrument toward you to minimize the formation of a wire ledge* (fig. 8-8).

5. *Lubricate the stone during sharpening.* (Stones are often immersed and stored in oil to ensure lubrication.)

*Black, G. V. Operative Dentistry, ed. 9. Milwaukee, Medico-Dental Publishing Co., 1955, vol. 2, p. 447.

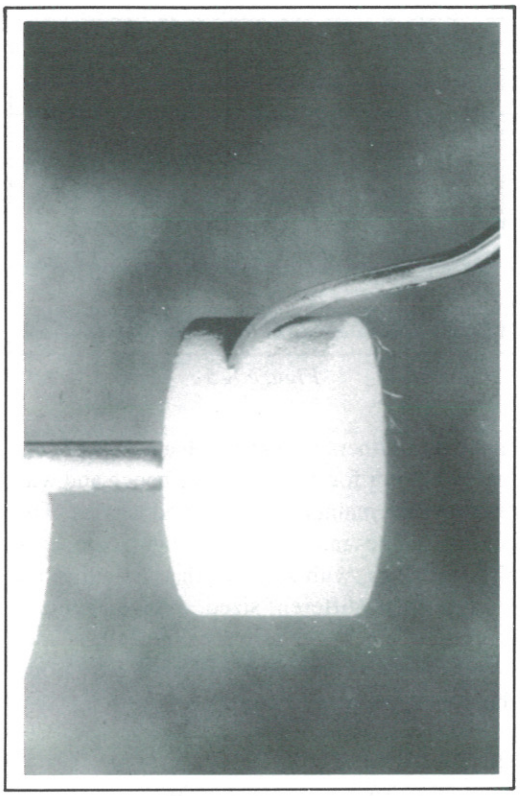


Figure 8-6

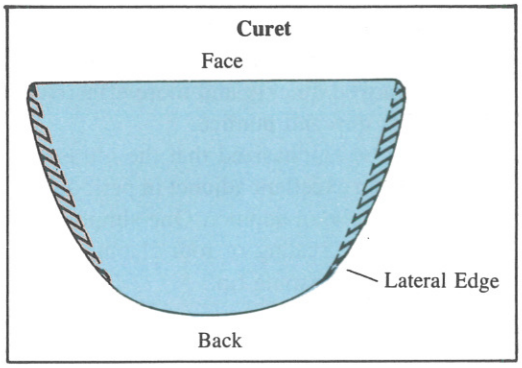


Figure 8-7

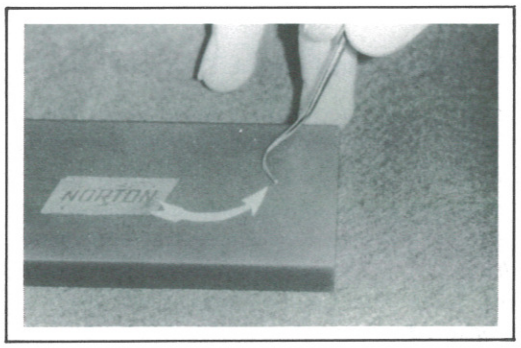


Figure 8-8

TEST FOR SHARPNESS

To determine whether an instrument has been properly sharpened, examine the cutting edge with a magnifying glass under good lighting. A sharp edge is a line, rather than a surface, and will not reflect light. A thin, bright line indicates a dull surface. As an instrument is sharpened, the bright line disappears. The fingernail method is not always a true test for properly sharpened instruments.

All hand instruments will require periodic sharpening. It is often necessary to resharpen an instrument during the treatment procedure. It is a good rule to sharpen instruments after each scaling and root planing before instrument sterilization.

Remember, if you scale and root plane with a dull instrument, you will waste time, work harder than you should, and have a poor end result.

Tooth movement in periodontal therapy

9

Principal Considerations
- Philosophy
- Root resorption
- Indications

Questions relevant to treatment
- Is minor tooth movement indicated?
- Should minor tooth movement be attempted?
- What method should be used?
  - Elastics
  - Bonded adhesive systems
  - Hawley appliance
  - Grassline ligature or elastic cord
- How much force should be applied?
- What type of retention will be needed?
- Results

Principal Considerations

The subject of tooth movement in periodontal therapy is too broad for adequate coverage in the *Syllabus*. Therefore, the objective of this chapter is to establish a philosophy of treatment, rather than a specific methodology. For additional information on this subject the reader is referred to the text *Minor Tooth Movement in General Practice,* by Hirschfeld and Geiger, 3rd ed., 1973, St. Louis, The C. V. Mosby Co.

PHILOSOPHY

The importance of minor tooth movement in both prevention and treatment of periodontal disease is well established in the literature. Orthodontic techniques are certainly not new, but periodontists as well as general practitioners have been slow to incorporate tooth movement techniques into their treatment plans. This is true even though a minor orthodontic procedure is often paramount in the preservation of a tooth (or perhaps of an entire dental arch), and a drastic compromise may have to be accepted if tooth movement is not considered. Fear of orthodontic procedures is perhaps a carryover from inadequate undergraduate training in this phase of dentistry. This fear is reinforced by the dental literature, where every discussion of tooth movement is accompanied by many warnings, while equally difficult techniques in other fields of dentistry are often discussed with little or no mention of danger.

ROOT RESORPTION

The possibility that root resorption may occur during tooth movement should not be viewed out of perspective. Many consider orthodontic treatment and root resorption to be almost synonymous. When extensive root resorption does occur during tooth movement, it is usually a result of orthodontic treatment continued over a period of several years. Minor tooth movement requires only a few weeks or months. Cementum is more resistant to resorption than is bone because of its relative avascularity. This makes it possible to move a tooth within the bone quite safely, provided gingival inflammation and other acute periodontal problems have been resolved. The chance of extensive root resorption is slight when appliances are used with properly controlled forces.

INDICATIONS

Some of the principal reasons for tooth movement are:

1. To place teeth in a healthier anatomic position for protection of the periodontium.
2. To correct tooth position and thus improve the function of the dentition.
3. To close an existing space where a tooth is missing and thus eliminate the need for a prosthetic replacement.
4. To enlarge a space or to improve the alinement of an abutment tooth for a prosthesis.
5. To return to their original position teeth that have drifted or fanned out because of periodontal disease and/or tongue-thrusting habits.
6. To move a periodontally involved tooth into an infrabony defect, thereby narrowing the width of the defect and improving the prognosis for treatment.
7. To improve esthetics. The importance of esthetics and the resultant attitude of the patient toward his natural dentition should not be overlooked. As children mature, their appearance becomes increasingly important to them. Many suffer years of unhappiness because their teeth are not attractive. When an individual is unhappy about the appearance of his teeth, their value is greatly reduced in his mind. This, in turn, affects the way he takes care of them and accounts for the gross neglect frequently observed in patients with unsightly dentitions.

Questions Relevant to Treatment

Before beginning tooth movement, the therapist should ask several questions relevant to treatment: Is minor tooth movement indicated? Should minor tooth movement be attempted? What method should be used? How much force should be applied? What type of retention will be needed?

IS MINOR TOOTH MOVEMENT INDICATED?

Severe congenital malocclusions and jaw deformities, such as prognathia, retrognathia, and apertognathia, should not be treated by minor tooth movement. The same applies to discrepancies in interarch relationships and severe malocclusions that have developed during the growth period. Minor tooth movement procedures are usually indicated when tooth malposition has been caused by such local factors as habits, periodontal disease, or drifting after extraction.

Other considerations, before minor tooth movement is undertaken, are as follows:

1. The patient must be cooperative and practicing effective plaque control.
2. There must be sufficient room for movement.
3. The periodontal and periapical prognosis of the tooth must be favorable.
4. If possible, all etiologic factors should be eliminated, even if fixed retention is planned.
5. The tooth must not be moved into a poor functional relationship, or into a relationship unfavorable to basal bone. For example, facial or lingual movement may result in dehiscences or fenestration of the alveolar cortical plate.
6. There must be enough teeth remaining for sufficient anchorage and retention.

SHOULD MINOR TOOTH MOVEMENT BE ATTEMPTED?

Motivation and plaque control must be thoroughly evaluated before tooth movement is begun. Treatment is contraindicated unless the patient is highly motivated and demonstrates effective hygiene.

The duration of the patient's assignment to the present duty station is also an important con-

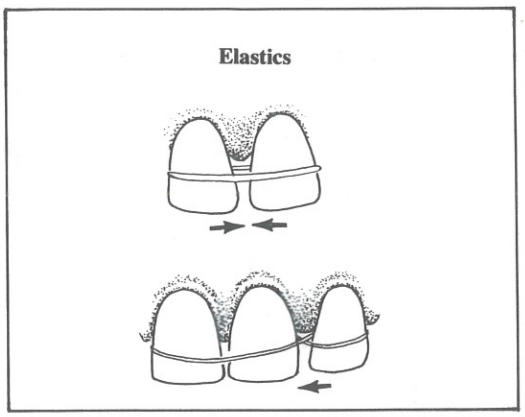


Figure 9-1

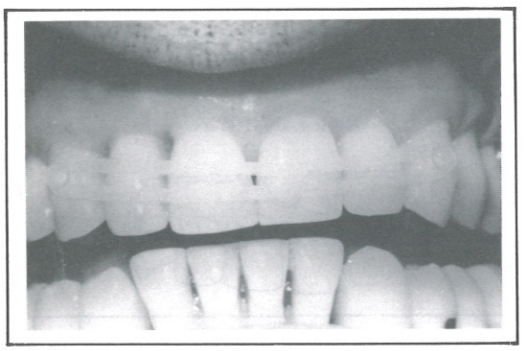


Figure 9-2

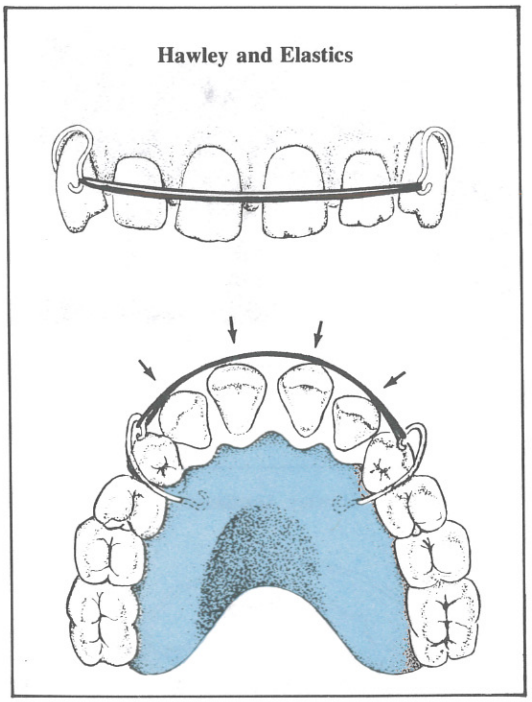


Figure 9-3

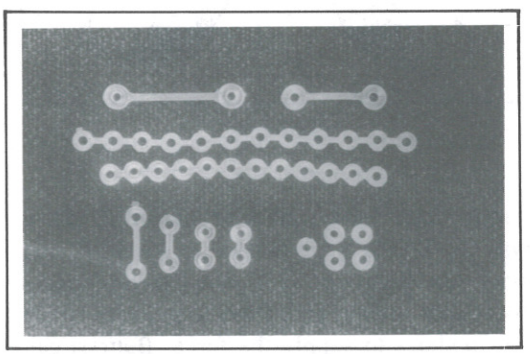


Figure 9-4

sideration. Some minor tooth movement procedures will require several months of active therapy and an additional period for retention. It is highly desirable that the same therapist begin and complete tooth movement.

WHAT METHOD SHOULD BE USED?

Various techniques and different types of materials may be used to apply the forces required for tooth movement. Some of the materials and methods currently used in minor tooth movement are discussed.

Elastics

Rubber elastics provide the simplest method of moving teeth together in a mesiodistal direction. The elastics can be used singly (fig. 9-1), in conjunction with a bonded adhesive system (fig. 9-2), or with a modified Hawley appliance (fig. 9-3) to move teeth in almost any direction. Extreme caution must be exercised when elastics are used singly. Severe periodontal destruction can result when elastics are accidentally displaced beneath the gingival tissue. The disposition of the elastics must be continually monitored and patients must be thoroughly instructed in their placement and daily management. Elastics offer the advantage of the application of uniform force to all teeth that they span. They are excellent for coaxing anterior teeth back into good alinement when there has been drifting and spacing.

Prefabricated orthodontic force modules afford the same advantage as rubber elastics but offer the additional advantage of a wide variety of sizes and shapes, which permits a somewhat more predictable application of force (fig. 9-4).

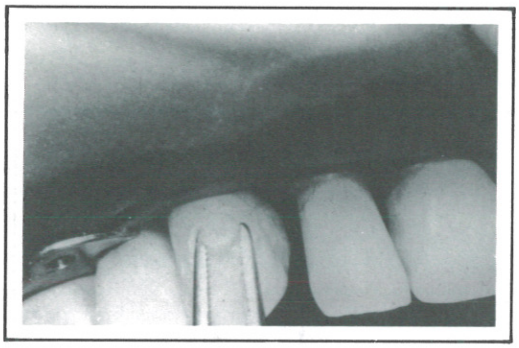


Figure 9-5

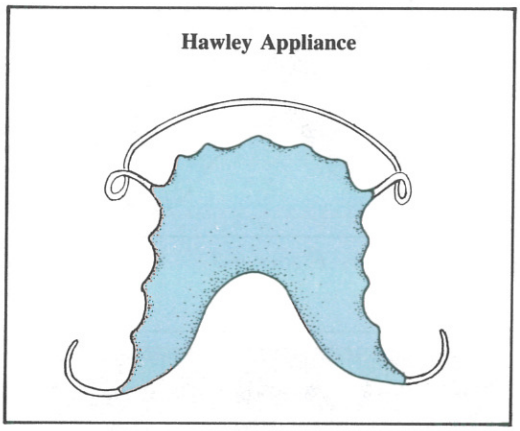


Figure 9-6

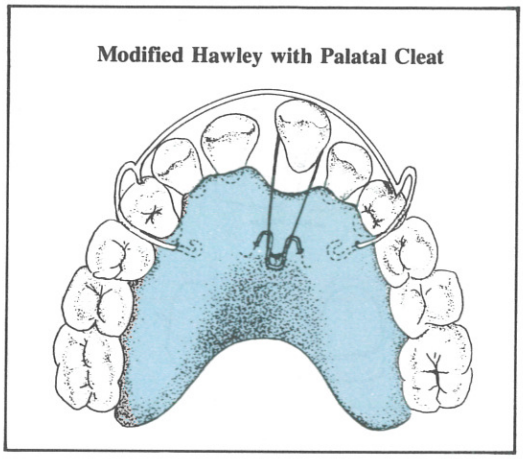


Figure 9-7

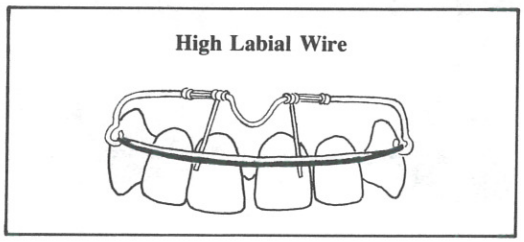


Figure 9-8

The modules work well with bonded adhesive systems. Brackets are bonded to the teeth, and tooth movement can be accomplished by using essentially straight arch wires to guide the teeth and modules to supply the force. Buttons can also be bonded to the teeth and the modules attached to the buttons. The modules retain much of their initial elasticity, even after several weeks in the oral environment. This is a decided advantage over rubber elastics, which must be replaced at least twice a day.

Bonded Adhesive Systems

Adhesive systems, which permit bonding of cleats, buttons, and brackets directly to the tooth surface, have added a new dimension to minor tooth movement (fig. 9-5). The bonded adhesives are particularly helpful for those who have never mastered banding techniques or who lack the equipment necessary to utilize metal bands. The various retainers are easily applied and have surprising retentive capacity. The adhesive systems are versatile and are designed to accommodate techniques that incorporate arch wires, elastics, modules, or combinations thereof. The bonding material is readily removed with minimal loss of tooth structure. It also serves as an excellent material for splinting and retention, after orthodontic movement.

Hawley Appliance

The Hawley is one of the oldest appliances used in dentistry (fig. 9-6). Originally designed as a retainer, it has since been modified for tooth movement. The modified Hawley appliance (fig. 9-7) and the high labial wire (fig. 9-8) are particularly useful for moving teeth in a palatal or a lingual direction.

The first step in designing a Hawley appliance is to determine the desired movement, and to incorporate the various kick springs, cleats, bite planes, elastic hooks, etc., in the design (fig. 9-9). The acrylic resin should be carried 1 to 2 mm onto the lingual surface of the

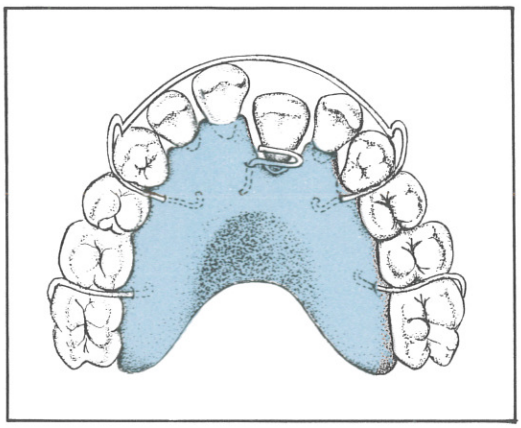


Figure 9-9

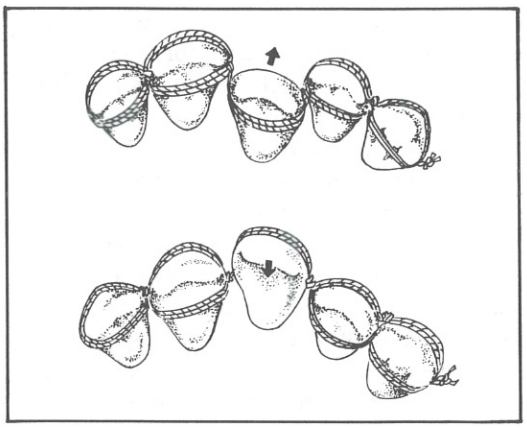


Figure 9-10

supporting teeth for stabilization. A horseshoe palate design is preferable to full palatal coverage. Retention of the appliance is derived from at least two clasps, placed preferably on the first molars.

The anterior labial arch wire of the Hawley appliance is usually 0.030 chrome alloy wire or 0.036 gold wire. A high labial arch must be thicker (0.040) to reduce its flexibility. Preformed labial arch wires may be obtained commercially. Usually 0.022 chrome alloy is used for labial and lingual kick springs. The patient is instructed to wear the appliance day and night, except during meals and during plaque control procedures. Adjustments are usually made at 1-week intervals.

Grassline Ligature or Elastic Cord

Grassline ligatures or elastic cord are excellent for moving teeth short distances somewhat rapidly. Patients should be seen at 48-hour intervals to assess comfort and tooth movement, and to replace the ligature if necessary (fig. 9-10).

HOW MUCH FORCE SHOULD BE APPLIED?

The less force used in moving teeth, the less chance there is that the procedure may cause damage. Too much force results in overcompression of the periodontal ligament with resultant necrosis and delayed movement. Generally, light pressure should be used over a long period when a tooth is to be moved a considerable distance. Heavy pressure may be used for a short period when a tooth is to be moved a very small distance. Light force is produced by rubber elastics, medium force by the elasticity of metal (Hawley appliances), and heavy force by twisted wire ligatures. Whenever possible, the force placed on a tooth should be measured. A Dontrix stress gauge can be used for this purpose. The range of force is as follows:

Light force—2 to 4 ounces (60 to 120 grams)

Medium force—4 to 6 ounces (120 to 180 grams)

Heavy force—over 6 ounces (180 grams or more)

It is often difficult to accurately gauge the pressure exerted by some appliances. In any case, the patient himself is a good indicator of the degree of force that can be tolerated. He should be alerted to expect some soreness of the tooth for 24 to 72 hours, but that severe or persistent pain after this time is a danger signal. He should be instructed to return immediately if the pain is intense, or to remove the appliance until an adjustment can be made.

WHAT TYPE OF RETENTION WILL BE NEEDED?

Once the teeth are repositioned, the problem is one of retention. This is the period when the periodontium reorganizes. Ideally, retention should be required only during the time needed for the periodontal fibers to reorganize to a new position on the tooth, and for the bone to mature into normal alveolar bone. Retention can be accomplished by means of a bite guard appliance, bonded acrylic resins, wire and acrylic resin splints, or a Hawley retainer. Long-term use of Hawley retainers is generally contraindicated.

These appliances are potentially destructive to the periodontium and, if used for long-term retention, may contribute to periodontal traumatism, recession, and generalized periodontal breakdown. If it is impossible to eliminate all etiologic factors, or if tooth mobility is pronounced, a permanent type of splint should be considered. Teeth that have been moved from a cross-bite to a normal occlusal relationship rarely need retention. The occlusion should be carefully checked and adjusted following tooth movement, and rechecked from time to time during the retention period. Most teeth that have undergone minor movement should be retained for 2 to 6 months. Teeth that have been rotated may require a longer period, unless periodontal surgery is performed immediately after they have been rotated. Severing of the gingival fiber apparatus appears to reduce the opportunity for relapse and also the period required for retention.

RESULTS

The rewards of minor tooth movement in periodontal therapy are numerous. One has only to see the dramatic positive change in patient attitude, personality, and appearance to realize the importance of this treatment. Of equal significance, however, is the great potential for both prevention and management of periodontal disease. The severely malpositioned tooth of a young patient today is a potential periodontal problem of tomorrow.

Occlusion and occlusal adjustment

10

Occlusion
- Features of optimal occlusal function
- Prophylactic occlusal adjustment
- Periodontal traumatism
  - Primary periodontal traumatism
  - Combined periodontal traumatism
  - Secondary periodontal traumatism

Occlusal adjustment
- Objectives
- Odontoplasty
- Selective grinding
  - Centric prematurities
  - Protrusive prematurities
  - Working prematurities
  - Nonworking prematurities
- Prematurities, parafunction, and protection

Bite guards
- Indications
- Fabrication

Occlusion

Most practitioners do not approach the subject of occlusion with confidence, yet occlusion has been acknowledged by some as the great common denominator of all disciplines of dentistry. This unfortunate paradox has resulted from confusion and controversy surrounding the terminology, as well as from the concepts of occlusion. Rigid rules for occlusal adjustment and complicated occlusal gadgetry have been offered as substitutes for a more practical understanding of occlusion. Evidence of the confusion is seen every day as one observes occlusions that have been "overground"—frequently in the absence of any pathosis of occlusal origin. Conversely, many dentitions that require occlusal adjustment have not been treated. Unfortunately, clinical experience shows relatively few cases representing precise occlusal analysis followed by rational, accurate, and successful occlusal adjustment.

FEATURES OF OPTIMAL OCCLUSAL FUNCTION

Beyron* provides an excellent discussion of a concept of optimal occlusion, which may be summarized as follows:

1. Optimal occlusal function denotes an absence of cuspal interference on closure and during contact gliding movement.

2. It has bilateral distribution of load on a large number of teeth in the intercuspal position and on a reasonable number of teeth in the retruded contact position. The distance between these positions should be small—about 1 mm. This distance should be traversed smoothly and without interferences, a possibility more easily

*Beyron, H. Optimal Occlusion, Dent. Clin. North Am. 13:537–554 July 1969.

realized if these two positions are located on the sagittal plane.

3. Chewing should be possible with equal ease on right and left sides, facilitated by simultaneous gliding contacts between the teeth on the working side—group function.

4. Optimal occlusion would have axial loading of teeth with forces directed within the supporting bone area.

5. It should have an acceptable interocclusal distance.

6. In general, optimal occlusion should minimize the likelihood of bruxism or other potentially destructive parafunctional movements.

Obviously, patients often exhibit many variations from these criteria and do not suffer occlusal injury. This is because of the adaptive capacity of the neuromuscular system, temporomandibular joints, periodontal tissues, and teeth. These occlusions are adequate and usually require no adjustment. The features of an optimal occlusion are valuable as guidelines to occlusal therapy. An occlusion should not be considered pathologic unless tissue injury resulting from occlusal stress can be demonstrated.

PROPHYLACTIC OCCLUSAL ADJUSTMENT

If the therapist appreciates the universal importance of occlusion, he will examine the occlusion on every patient. There is a common tendency to examine, document, and treat only those cases in which the occlusal factor is so prominent that it cannot be ignored. The admonition to become more occlusion-conscious is not a plea for "prophylactic occlusal adjustment." Indeed, it is definitely worse to overtreat occlusion than to ignore this area completely.

Ideally, the dentist should strive to develop his own "positive" occlusal interest while establishing or preserving his patient's "negative" occlusal awareness. In other words, he must strive to establish an occlusion so functional, physiologic, and comfortable that the patient doesn't notice it. This is true "harmony within the stomatognathic system."

Again, the decision to treat or not to treat occlusion should be based entirely upon the existence of periodontal traumatism, undesirable occlusal relationships (plunger cusps, uneven marginal ridges, etc.), temporomandibular joint dysfunction, and muscular disorders that are related to occlusal activity. Such symptoms may be indicative of "pathologic occlusion."

PERIODONTAL TRAUMATISM

The therapist finds himself in a semantic jungle when considering the tissue damage resulting from occlusal forces that exceed the adaptive capacity of joints, muscles, or periodontium. For simplification, the following definitions will be used:

1. *Traumatogenic occlusion:* excessive force.

2. *Trauma from occlusion:* the lesion produced in the tooth-supporting tissues by this force.

3. *Periodontal traumatism:* the entire process, outlined in Table 10-1.

Periodontal traumatism may be divided into three general categories: primary, combined, and secondary.

Primary Periodontal Traumatism

Primary periodontal traumatism is usually a result of excessive force, associated with such factors as parafunctional habits, high restorations, removable partial dentures that traumatize abutment teeth, and drifting of teeth following extraction. There is no pocket formation, and the lesion can usually be corrected by eliminating the local etiologic factor and/or adjusting the occlusion. Figure 10-1 illustrates the lesions produced in primary periodontal traumatism by excessive force acting on a healthy periodontium. Note the location of the lesions and the variation in width of the periodontal ligament space as the tooth is moved around its center of rotation.

Combined Periodontal Traumatism

Combined periodontal traumatism is the result of excessive force on a diseased periodontium. In effect, inflammation is present; some pocket formation has occurred; and excessive force, generally from parafunctional movements, can exaggerate the disease process. Figure 10-2 illustrates the lesions produced in combined periodontal traumatism by excessive force on a diseased periodontium. In this instance, traumatogenic occlusion is a co-destructive factor in combination with existing inflammation. The lesion produced is not reversible by occlusal adjustment alone.

TABLE 10-1. PERIODONTAL TRAUMATISM

Trauma from occlusion	Etiology	Pocket formation	Reversible by occlusal adjustment	Crown to root ratio	Splinting
Primary	Excessive force	No	Yes	Normal	No
Combined	Usually excessive force and inflammation	Yes	No (inflammatory lesion also requires control)	Increased	Sometimes
Secondary	Usually normal forces	No		Severely increased	Sometimes

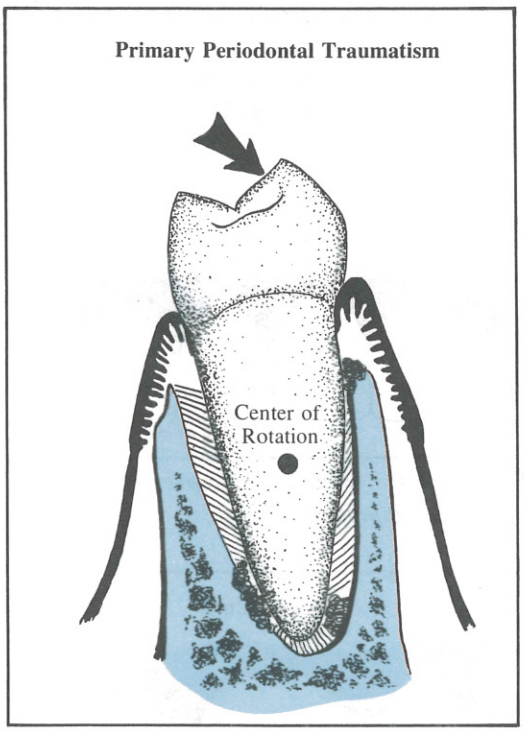


Figure 10-1

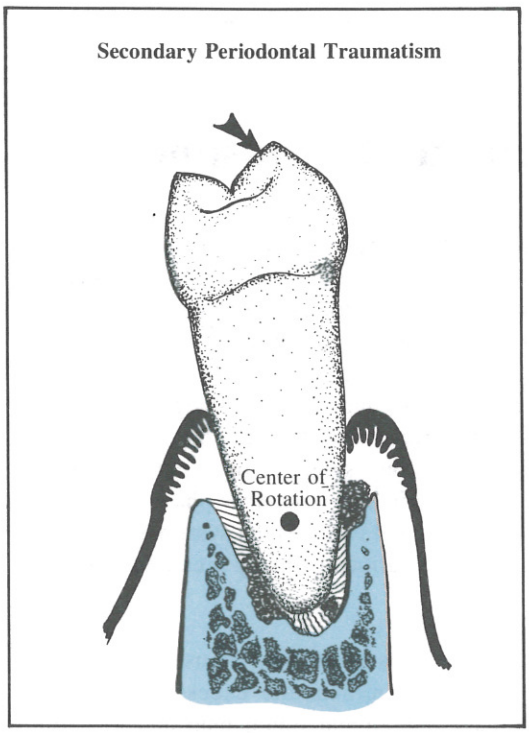


Figure 10-2

Secondary Periodontal Traumatism

Secondary periodontal traumatism is the result of normal occlusal forces on a severely weakened periodontium as illustrated in figure 10-3. There is a marked increase in crown-to-root ratio, and splinting is sometimes necessary. Secondary periodontal traumatism is often observed following treatment of advanced cases of chronic destructive periodontitis. The gingival tissues are restored to health, but the periodontium is so weakened as a result of the disease process that normal occlusal forces result in tooth mobility.

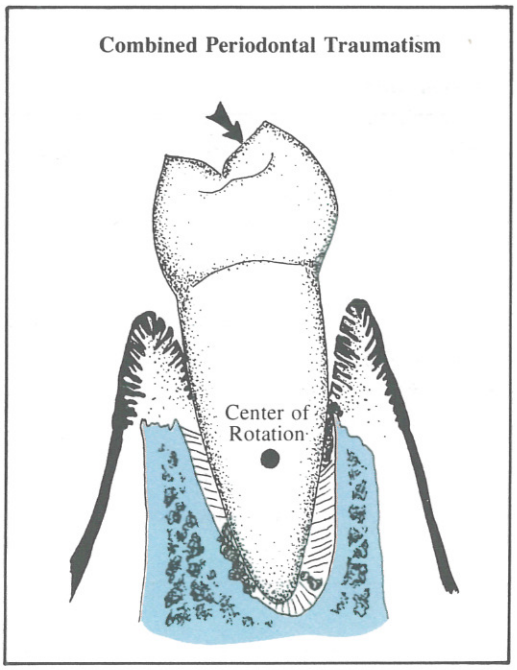


Figure 10-3

Occlusal Adjustment

Occlusal adjustment of the natural dentition may be defined as a method of reshaping teeth that are producing injury to other teeth or to the supporting structures. Odontoplasty is the reshaping of teeth to eliminate *gross* occlusal relationships and contours (plunger cusps, sharp incisal edges, marginal ridge relationships, etc.) and may be considered one stage of occlusal adjustment. Selective grinding is another phase of occlusal adjustment and includes the systematic elimination of the various pathway prematurities.

OBJECTIVES

The objectives of occlusal adjustment are:

1. To eliminate the excessive forces of primary periodontal traumatism and to permit healing of the attachment apparatus.
2. To eliminate or reduce the forces that exaggerate the disease process in periodontitis.
3. To promote gingival health by restoring form and function; e.g., by creating contours that will reduce plaque accumulation, eliminate food impaction, etc.
4. To help control parafunction by elimination of obvious locking or deflective contacts, and by elimination of high restorations that encourage clenching, grinding, and doodling habits.
5. To improve esthetics and the functional requirements of patients; e.g., by smoothing ragged incisal edges, which are unsightly and potentially injurious to lips, cheeks, etc.
6. To preserve a negative sense of occlusion. An induced dental awareness, or positive occlusal sense from occlusal adjustment, can result in extreme emotional anxiety and distress.

ODONTOPLASTY

Odontoplasty procedures have been advocated as initial steps to total adjustment, whenever such treatment is warranted. Correction of the undesirable occlusal relationships may, indeed, constitute most of the therapy.

The indications for reshaping the teeth are varied and extensive. Some of the more commonly encountered problems effectively treated by odontoplasty include:

1. Extruded teeth (fig. 10-4).
2. Plunger cusps (fig. 10-5).

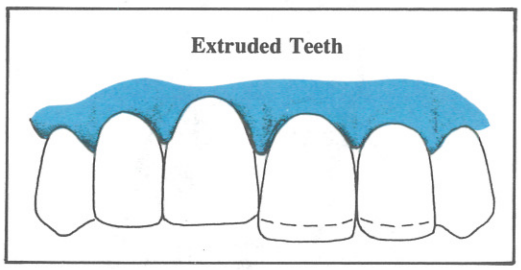


Figure 10-4. Dotted lines represent tooth removal by odontoplasty.

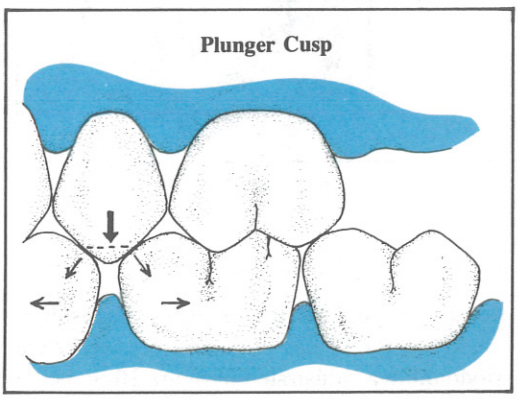


Figure 10-5. Dotted line represents tooth removal by odontoplasty.

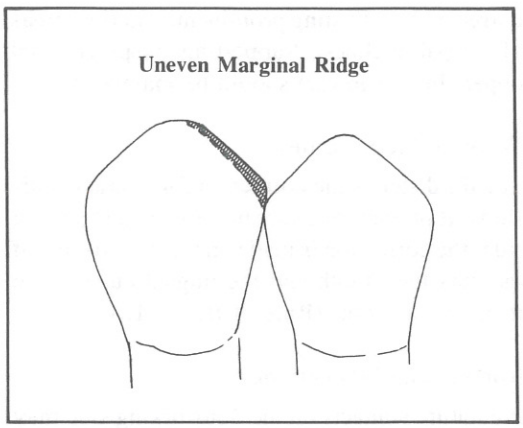


Figure 10-6

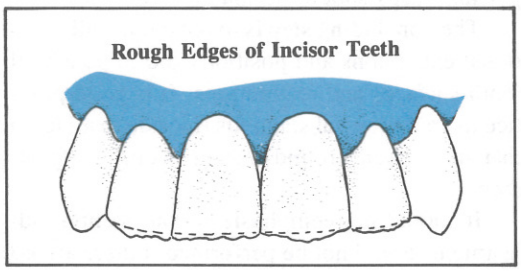


Figure 10-7. Dotted lines represent tooth removal by odontoplasty.

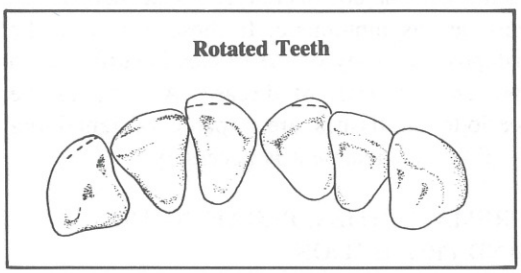


Figure 10-8. Dotted lines represent tooth removal by odontoplasty.

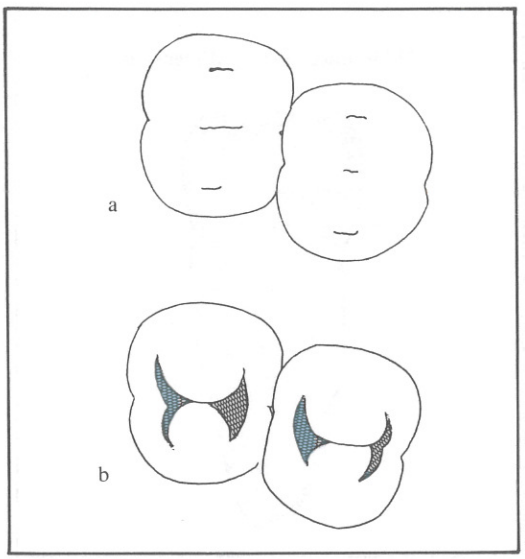


Figure 10-9. Shaded area represents site of odontoplasty.

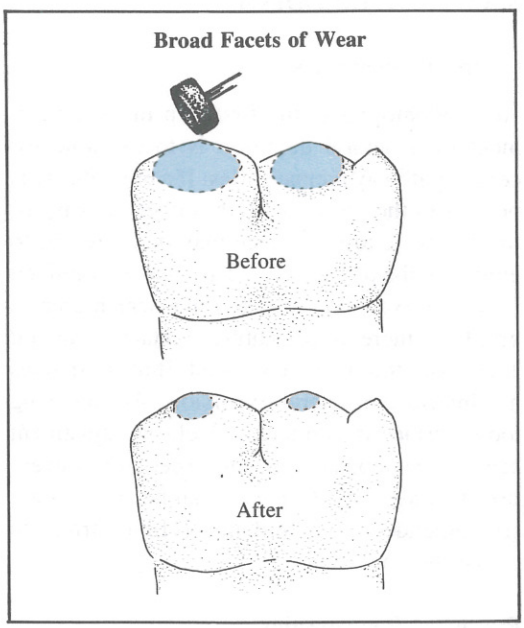


Figure 10-10

3. Uneven adjacent marginal ridges (fig. 10-6).

4. Fragile and rough edges of incisor teeth (fig. 10-7).

5. Rotated teeth (fig. 10-8).

6. Rough or poorly contoured restorations or marginal ridges (fig. 10-9).

7. Broad facets of wear (fig. 10-10).

8. Wide buccolingual dimension (fig. 10-11).

These conditions may be improved by judicious grinding to prevent wedging and impaction of food, to direct occlusal forces axially, to improve esthetics, to make the dentition easier to cleanse, and to establish the plane of occlusion prior to restorative procedures. In many cases, premature centric pathway contacts may be adjusted automatically by odontoplasty.

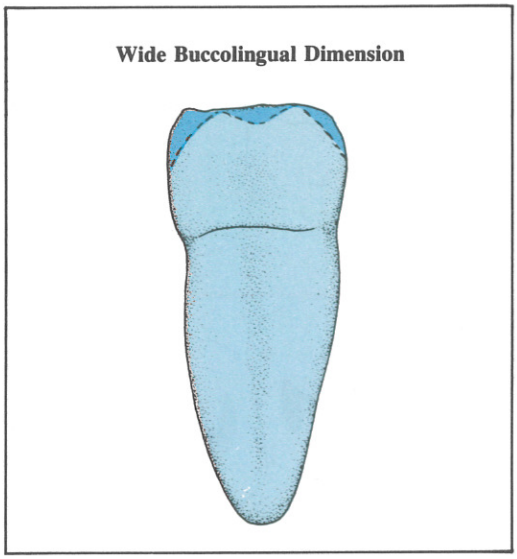


Figure 10-11

SELECTIVE GRINDING

Centric Prematurities

After odontoplasty, the first step in the adjustment of a natural dentition is to examine for centric pathway prematurities. If a prematurity is present in the posterior teeth, either the cusp or the opposing tooth surface may be corrected to eliminate the prematurity. The cusp is examined in lateral excursion to determine which area to grind. If there is premature contact in lateral excursion, the cusp is corrected. If the cusp does not interfere in lateral excursion, the opposing tooth surface is corrected. Occlusal adjustment can be accomplished by high speed or conventional handpieces. Conventional handpieces are recommended when the dentist is first learning to adjust the occlusion.

Protrusive Prematurities

The second step is the correction of protrusive position and excursion. If the anterior teeth contact prematurely, the lingual surfaces of the maxillary incisors are corrected, reducing the inclination of the incisal guidance from the point of centric contact, incisally. In some instances, where there is a broad centric contact, the mandibular incisors may be corrected. In protrusive position, the incisal edges of the maxillary teeth are corrected to achieve group function. Prematurities occurring in the posterior teeth are corrected by adjusting prominently marked areas of cuspal inclines. Supporting cusps (lingual upper, buccal lower) should be maintained.

Working Prematurities

The third step is the correction for lateral excursions. If prematurities are present on the working side, the correction is made on the buccal cusp of the maxillary tooth and the lingual cusp of the mandibular tooth. (Rule of B.U.L.L.)

Nonworking Prematurities

Premature contacts on the nonworking side may be corrected by grinding the buccal incline of the maxillary lingual cusp or the lingual incline of the mandibular buccal cusp. Once again, supporting cusps should be maintained.

The concluding step is to reexamine all functional excursions and positions and to polish all tooth surfaces and restorations. It is good practice to apply topical stannous fluoride to all teeth that have been ground during occlusal adjustment.

It should be reemphasized that occlusal adjustment should not be performed if there are no signs or symptoms of periodontal traumatism, undesirable occlusal relationships, or temporomandibular joint syndrome. Many natural dentitions that do not conform to the ideal occlusion are asymptomatic. In these instances, the adaptive capacity of the tissues is sufficient to prevent the onset of disease. As long as the periodontal tissues are capable of remaining healthy, no treatment is necessary.

PREMATURITIES, PARAFUNCTION, AND PROTECTION

Many authorities on occlusion believe the true significance of terminal hinge/centric relation pathway prematurities lies in their potential to trigger bruxism or other parafunctional movements. Furthermore, it is generally agreed that most periodontal traumatism results from these excessive, prolonged, unconscious, or conscious, habitual forces rather than from normal functional contacts. While some cases of bruxism and clenching may be relieved by judicious occlusal grinding, success should not be anticipated in most situations. In cases of discomfort resulting from "high" restorations, however, dramatic relief is frequently afforded by occlusal adjustment. Such timely, localized therapy may

also prevent development of a parafunctional habit of strictly dental origin.

Many consider these highly prevalent occlusal habits to be normal mechanisms for release of tension. They are regarded as pathologic only when they result in periodontal, temporomandibular joint, or muscular damage, or in excessive tooth wear.

Frequently, despite occlusal adjustment, the origins of parafunctional movements are so deep-seated that only psychotherapy may resolve the habit. This approach may be undesirable, impractical, or unavailable. Reliance upon an occlusal bite guard appliance then becomes a valuable alternative approach to control parafunctional stress. These devices may serve to actually break the parafunctional habit. At least they minimize occlusal purchase areas, distribute forces over all teeth, and provide effective splinting.

Bite Guards

INDICATIONS

Bite guards serve many different purposes. For example, they are used:

1. To treat parafunctions (bruxing and clenching). The bite guard serves to minimize the injurious effects of bruxism until adjustment of the occlusion is accomplished, or until emotional tension or anxiety is alleviated or reduced. The guard will also prevent the extensive wear of the occlusal surfaces so frequently observed with bruxism.
2. To treat temporomandibular pain dysfunction syndrome (myofacial pain dysfunction syndrome). The rationale for use of the bite guard to treat this syndrome is the elimination of all occlusal interferences and the reduction of pain and muscular spasm.
3. To splint mobile teeth temporarily.
4. To provide orthodontic retention after minor tooth movement.
5. To prevent trauma to palatal tissue (e.g., deep overbite).
6. To test the adaptability of the patient to a decrease in interocclusal space prior to construction of "bite-raising" prostheses.
7. To prevent extrusion of unopposed teeth.
8. To manage anxiety and emotional stress due to positive occlusal sense.

FABRICATION

Several techniques are described for the fabrication of bite guards, and most of them require the services of a prosthetic laboratory. A technique has been devised that enables the clinician to make the impression, and to fabricate the guard using laboratory facilities.

The procedure is as follows:

1. Fabricate the bite guard for the dental arch that exhibits the greater tooth mobility, or the arch that has the greater number of remaining teeth. Make an alginate impression, and pour a cast with rapid-setting plaster (fig. 10-12).
2. Trim the cast to an oval shape to remove all sharp edges and permit an adequate vacuum seal. Make a hole in the center of the cast to improve adaptation of the resin material (fig. 10-13). Pencil a line at the height of contour of

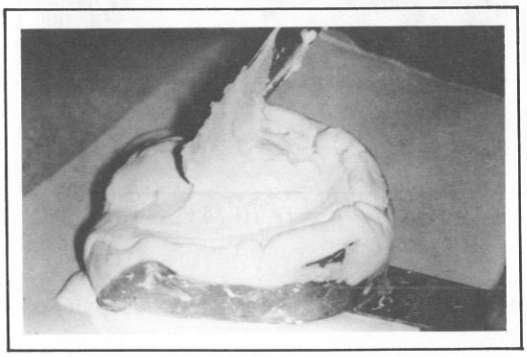


Figure 10-12

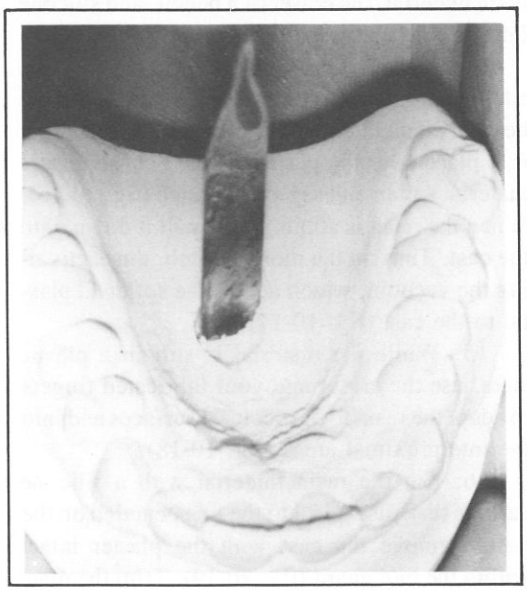


Figure 10-13

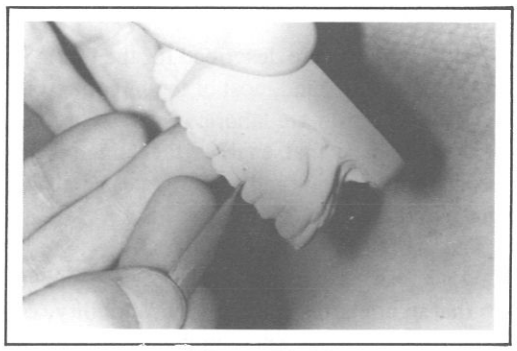


Figure 10-14

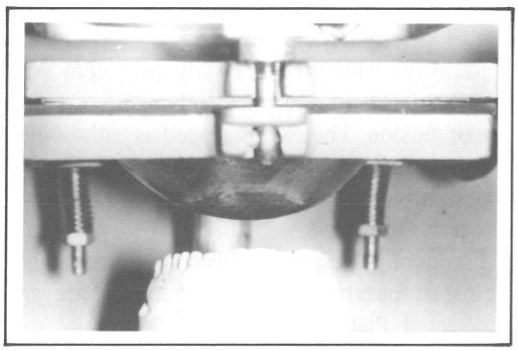


Figure 10-16

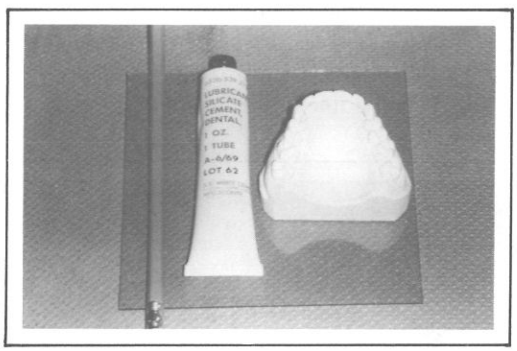


Figure 10-15

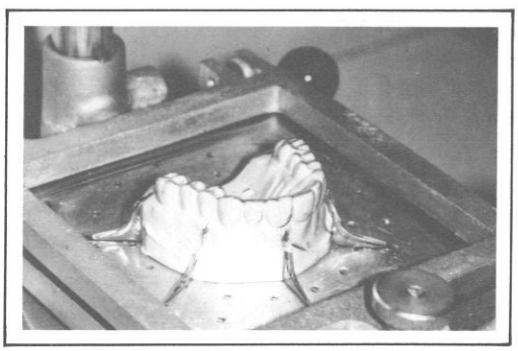


Figure 10-17

the teeth (fig. 10-14) and block out deep undercuts.

3. Adapt the bite guard material to the cast by means of a vacuum adapter. Use 0.060 clear resin material, the eraser of a pencil, and silicone lubricant to lubricate the fingers (fig. 10-15).

4. Place a sheet of resin in the vacuum adapter and secure with the locking nut. Turn the heating element on with the switch, and swing it into place over the plastic sheet. Let the plastic material soften and sag about 1 inch (fig. 10-16). When the resin is at this stage, pull it down onto the cast. Turn on the motor switch, thus activating the vacuum, which adapts the softened plastic to the cast (fig. 10-17).

5. While the material is still in a plastic state, use the eraser and your lubricated fingers to adapt the resin to the occlusal surfaces and into the interproximal areas (fig. 10-18).

6. Cut the resin material with a silicone carbide disk just apical to the line penciled on the cast. Remove the cast with the plaster intact within the bite guard (fig. 10-19). Trim the margin to a knife-like edge with a denture trimming

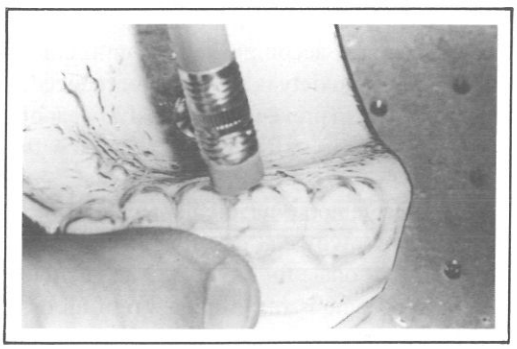


Figure 10-18

bur (Federal Stock Number 6520-076-8682) (fig. 10-20). Use a polishing wheel to finish (fig. 10-21). Then remove the plaster from within the bite guard and thoroughly clean the bite guard. Try it for fit.

7. Use rapid setting acrylic resin to fabricate an occlusal table. Moisten the occlusal surface of the bite guard with acrylic resin monomer to enhance the union of the acrylic resin to the

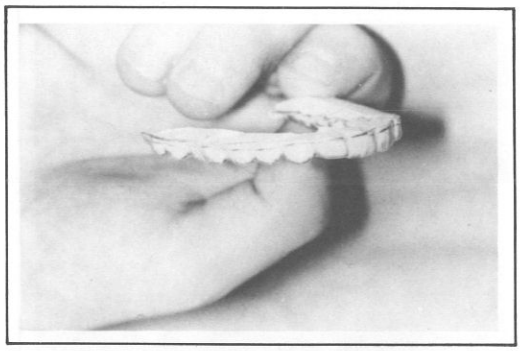


Figure 10-19

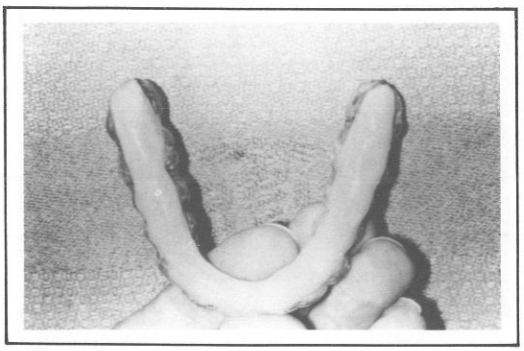


Figure 10-22

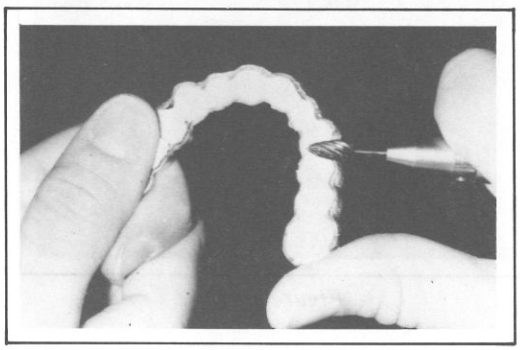
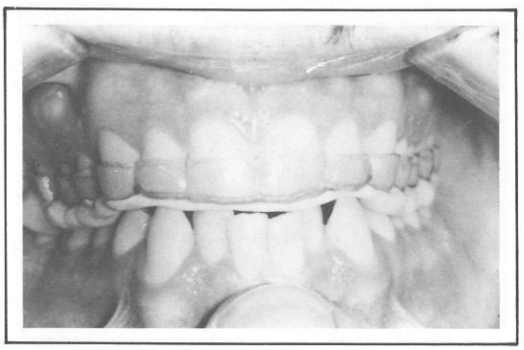


Figure 10-20



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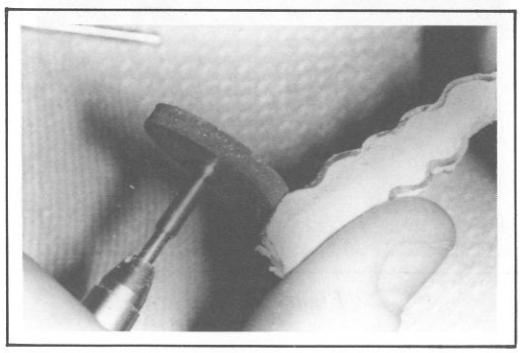

Figure 10-21

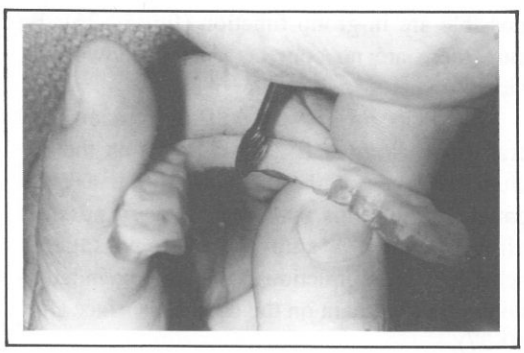

Figure 10-24

guard. Mix the acrylic resin in a dappen dish and place it on the occlusal surface of the bite guard (fig. 10-22).

8. Insert the guard into the patient's mouth. Guide his mandible into centric relation and instruct him to tap into the soft resin material lightly. Then instruct him to tap lightly in the position of habitual or functional occlusion (fig. 10-23).

9. Remove the guard and immerse it in warm water for 2 minutes to reduce the setting time of the resin. Trim the resin to remove the excess and to reduce the width of the occlusal table (fig. 10-24).

10. To adjust the bite guard, use articulating ribbon until maximum contact is achieved over the long axis of the teeth (fig. 10-25). Adjust the guard in working position until as many teeth as

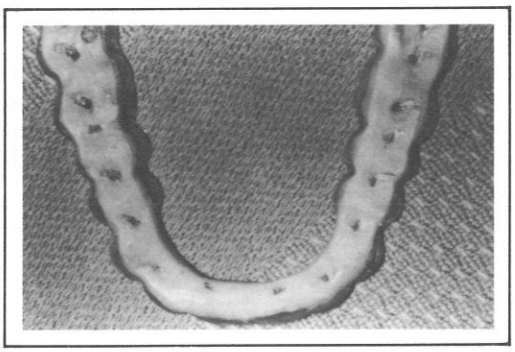

Figure 10-25

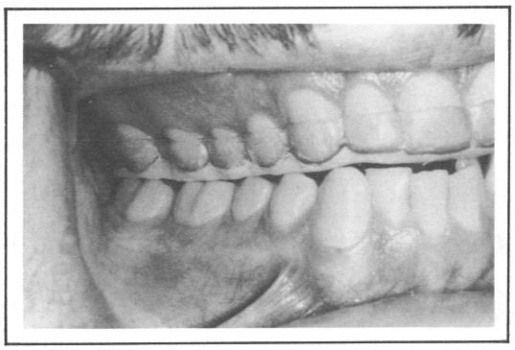

Figure 10-26

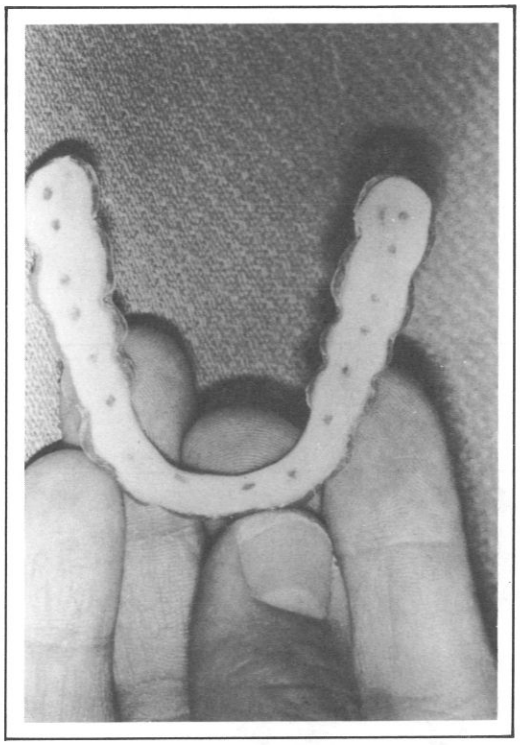

Figure 10-27

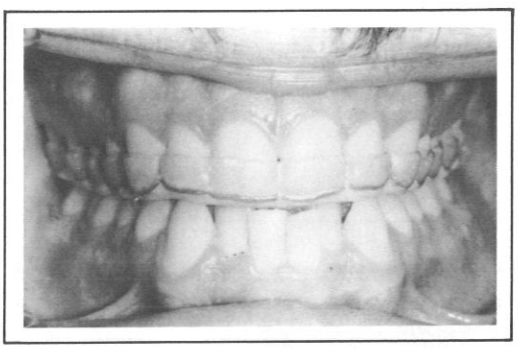

Figure 10-28

possible are in group function (fig. 10-26). Be sure there are no contacts on the nonworking side.

11. Place milling and polishing cream (Federal Stock Number 6520-500-0400) on the occlusal surfaces of the bite guard, and ask the patient to glide his mandible in every possible direction until a smooth, even passage is achieved. On completion of this step, no indentations should remain on the occlusal surface (fig. 10-27).

12. Lightly polish the guard and insert it for final fitting (fig. 10-28). The patient should be recalled in 3 to 4 days to verify the accuracy of fit and to correct any occlusal discrepancies that are noted.

In certain cases, adequate adjustment of the occlusion is not possible because of pain, muscle spasm, or trismus. It is recommended that resilient bite guard material be used, thus negating the need for adjustment of the occlusion. The resilient material has been found to achieve the desired results but wears more rapidly than the hard resin materials, and is considerably bulkier. In addition, patients cannot eat with the resilient material in place but manage amazingly well with the harder resin material.

11 Temporary splinting of teeth

Rationale for temporary splinting
  Criteria for selection of splint
Techniques for temporary splinting
  Anterior teeth
    Direct adhesive bonding system
    Circumferential wire ligature
    Intracoronal wire and acrylic resin splint
  Posterior teeth
    Intracoronal amalgam, wire, and acrylic resin splint
  Bite guard

A splint is an appliance used to stabilize or immobilize loose teeth. All the various types of permanent or temporary splints used in dentistry today, and the reasons for their use, will not be discussed in this chapter. It is important, however, to present some rationale for temporary splinting and a few of the available techniques.

Before the therapist decides to fabricate a splint of any type, he must weigh the advantages against the major disadvantage of most splints—the compromising of plaque control. Generally, when teeth are splinted the patient must work harder and longer to accomplish the same level of cleanliness. Many patients are unwilling, others unable, to spend the extra time required to effectively cleanse their teeth under such a handicap. Consequently, techniques should be employed which do not seriously jeopardize the cleansing of the interproximal space.

Rationale for Temporary Splinting

Before the various temporary splints are discussed, it is important that the reasons for splinting be explained. They are as follows:

1. *To assist in the healing of mobile teeth following periodontal treatment procedures.* Excessive tooth mobility may disrupt and retard healing.

2. *To determine whether teeth with a borderline prognosis should be retained.* If stability is not achieved after 2 to 3 months, no further improvement can be expected, and extraction is indicated.

3. *To reduce discomfort and stabilize teeth involved by a periodontal abscess.*

4. *To stabilize teeth that have been loosened by extensive force.* The teeth may have been damaged by an athletic injury or an automobile accident.

5. *To retain teeth in position following orthodontic movement.*

CRITERIA FOR SELECTION OF SPLINT

There are many different types of temporary splints. The therapist should select the one that will:

1. Distribute occlusal forces over as many teeth as possible.
2. Prevent the migration of teeth.
3. Not irritate the cheeks, lips, tongue, or gingival tissue.
4. Permit the patient to attain effective plaque control.
5. Result in the least amount of removal of tooth tissue.
6. Be accepted esthetically by the patient.
7. Be easily and economically prepared.

Techniques for Temporary Splinting

The following techniques are considered applicable to the practice of periodontics and generally fulfill the aforementioned criteria for a temporary splint.

ANTERIOR TEETH

Direct Adhesive Bonding System

This method is very effective in stabilizing mobile teeth and may prove to be *the* temporary splint of the future. It is easy to apply, is nonirritating to oral tissues, is esthetic, and requires minimal removal of tooth tissue. Most important, it allows patients to readily accomplish plaque control. Experience has shown that the direct bonded splint will remain intact at least a year. One word of caution—direct bonding adhesive may fuse with acrylic resin restorations. Also, the material does not bond well to ceramics. Figure 11-1 demonstrates one stage of fabrication of a direct bonded splint that utilizes ultraviolet light to polymerize the resin. This technique has the advantage of unlimited working time, because polymerization does not occur until the resin is exposed to the ultraviolet light. This allows adequate time for proper placement and manipulation of the resin in the interproximal spaces. Composite resins of this type have been shown to have good compressive strength, high resistance to fracture, and minimal marginal leakage; however, they are subject to abrasion. Studies have also shown that smoking and the use of phenol- or formaldehyde-containing toothpastes may lead to disintegration of these materials.

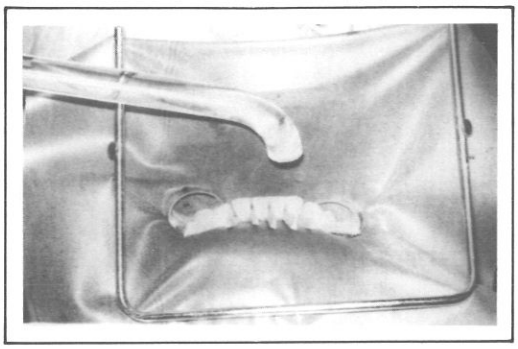

Figure 11-1

Circumferential Wire Ligature

This ligature is a widely used form of temporary stabilization. Its use is limited to anterior teeth and, unless carefully placed, it can seriously interfere with the patient's plaque control procedures and can move teeth out of proper alinement. It is applied as follows:

1. A double circumferential loop of wire (0.01 inch, dead-soft) is positioned horizontally over the teeth, encompassing the mobile teeth and a sufficient number of firm teeth (fig. 11-2). A single loop of thicker wire (0.02 inch) may also be used. After the facial and lingual sections of the wire have been adapted to the teeth, the free ends are twisted together and tucked into an interproximal space.

2. Short sections of wire (0.01 inch, dead-soft) are then inserted interproximally, engaging the facial and lingual sections of the circumferential wires (fig. 11-3), and are twisted.

3. The excess wire is trimmed (fig. 11-4), and the twisted ends are tucked into the interproximal spaces (fig. 11-5).

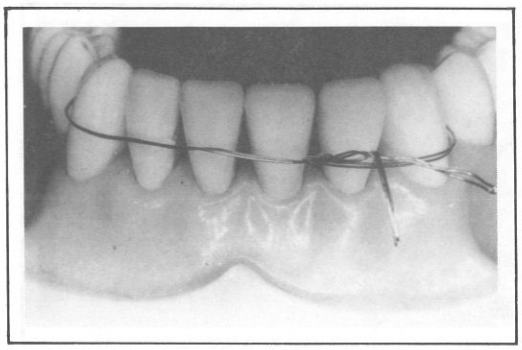

Figure 11-2

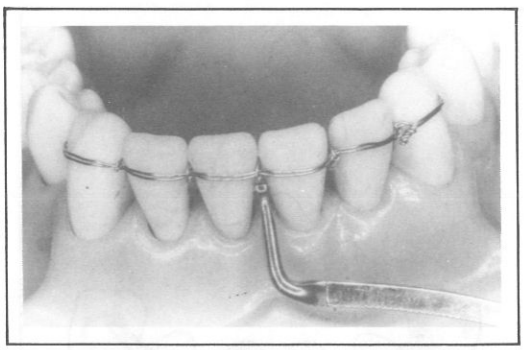

Figure 11-5

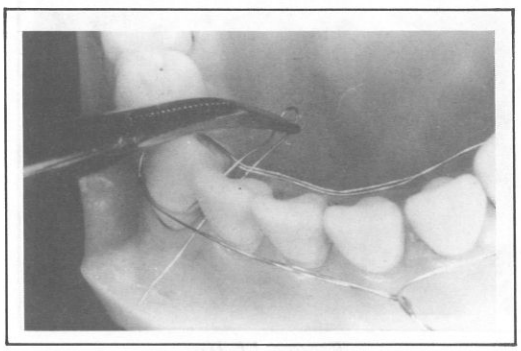

Figure 11-3

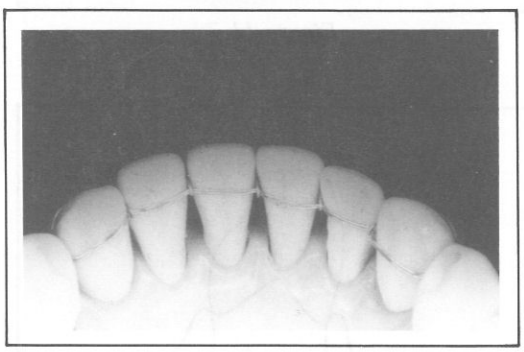

Figure 11-6

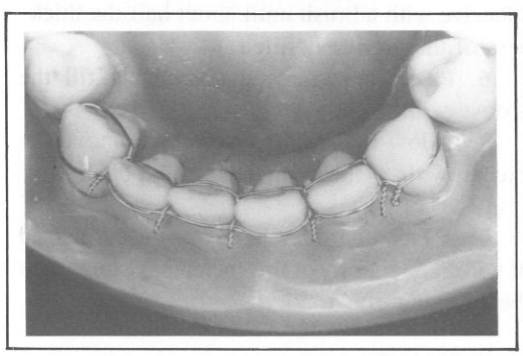

Figure 11-4

4. The circumferential wire must be positioned coronal to the cingula and gingival to the contact areas to prevent the ligature from being displaced in a gingival or an incisal direction (fig. 11-6).

5. The entire splint is often covered with self-curing acrylic resin. This material increases the strength and longevity of the splint while making it more esthetic and less of an irritant to the patient's lips and tongue.

Intracoronal Wire and Acrylic Resin Splint

These splints were originally designed to be used for short periods of time. Experience has shown, however, that they can function for several years, if so desired. The technique for fabrication is as follows:

1. Prepare an undercut slot the width of a No. 559 bur and about 1.0 to 1.5 mm deep, midway between the cingulum and the incisal edge of the teeth.

2. Measure a length of Markley wire, or braided 0.010 inch dead-soft wire, the full length of the channel.

3. Apply rubber dam.

4. Place Stim-U-Dents or endodontic points interproximally to limit the flow of resin.

5. Half fill the channel with self-curing acrylic resin, and lay the wire to place (fig. 11-7).

6. Apply additional acrylic resin to the height of the preparation.

7. Allow the resin to harden. Remove the excess with a finishing bur, and polish (fig. 11-8).

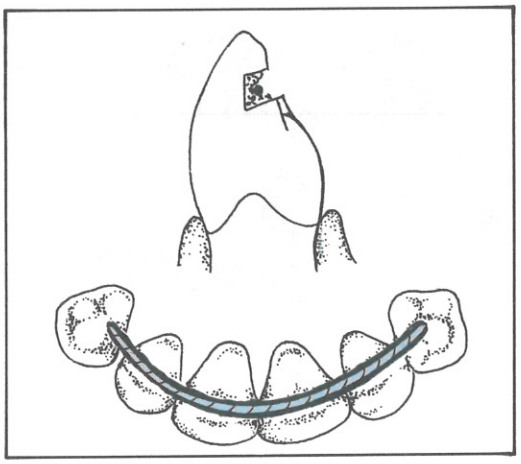

Figure 11-7

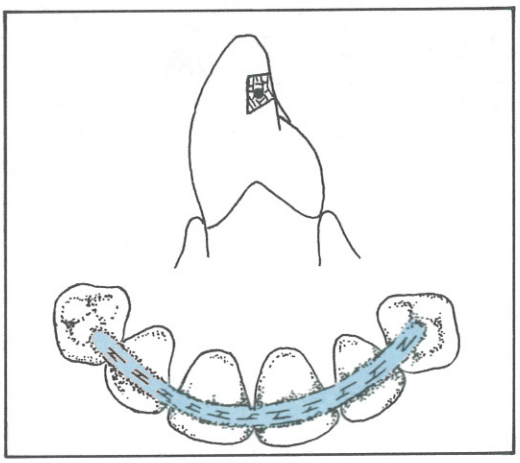

Figure 11-8

POSTERIOR TEETH

Intracoronal Amalgam, Wire,
and Acrylic Resin Splint

This technique is similar to the intracoronal technique described for anterior teeth. The splint has also been shown to function for long periods of time without breakage or fear of dental caries. The technique is as follows:

1. Prepare a simple undercut groove in amalgams of the involved teeth. It should be about 3 mm wide and 1.5 mm deep.
2. Measure a length of Markley wire, or braided 0.010 dead-soft wire, to fit the entire length of the groove (fig. 11-9).
3. Apply rubber dam.
4. Place Stim-U-Dents or endodontic points interproximally to limit the flow of resin.

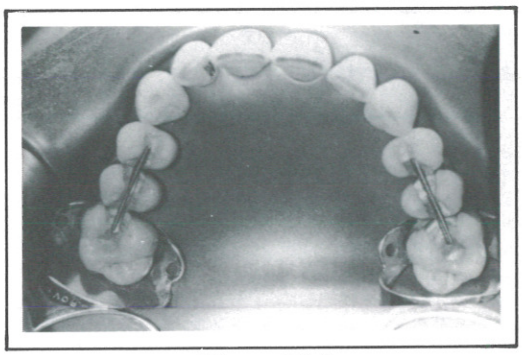

Figure 11-9

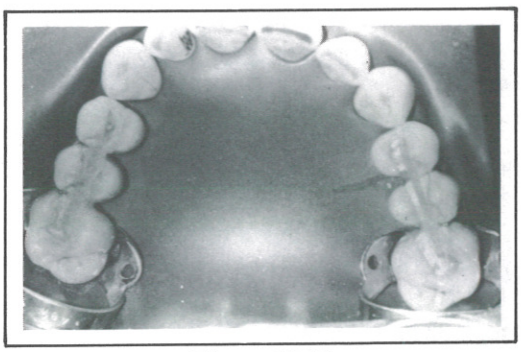

Figure 11-10

5. Apply self-curing resin or direct bonding adhesive with a brush until about half the thickness of the groove is filled.
6. Place the wire and completely fill the groove with resin.
7. Allow the resin to harden. Remove the excess with a finishing bur, and polish (fig. 11-10).

Cast Ticonium bars have been used instead of wire. The rigidity of this metal may strengthen the splinting effect.

BITE GUARD

This is an excellent temporary splint for anterior and posterior teeth. It distributes occlusal forces over all remaining teeth in the arch, is esthetic, does not require removal of tooth tissue, and can be removed by the patient while cleansing the teeth. The guard can be worn 24 hours a day and the patient can eat with it in place. Some patients experience difficulty in speaking clearly at first, but with practice (reading out loud) this problem can be quickly overcome. Patient acceptance for the bite guard is high, even when worn 24 hours a day. See chapter 10 for details on fabrication.

Wound healing 12

Healing of soft tissue wounds
- Excision of gingiva
- Simple incision
- Reattachment
- New attachment
- Open wounds
- Healing of flaps
- Healing of free gingival grafts (free soft tissue autograft)
- Healing following scaling and root planing

Healing involving bone
- General principles
- Infrabony defects
- Osseous autografts
  - Fate of autografts

Other considerations in wound healing
- Nutrition and systemic disorders
- Age
- Asepsis
- Healing rate

Wound healing applied to periodontal surgery

All surgery implies a tearing asunder of the existing relationship of various cells and tissues of the body. Healing is the restoration of these body elements into a new physiologic and anatomic relationship. It generally includes all of the following: clotting, granulation, epithelization, collagen formation, regeneration, and maturation.

An understanding of the healing process will permit the operator to design and execute a rational surgical procedure to support his overall therapeutic objectives. It will also ensure that his intention is accomplished and that the patient's healing period is as brief and comfortable as possible.

Periodontal surgery can be broadly classified into two categories: Procedures designed to correct soft tissue defects, and those that deal with the elimination of hard tissue defects.

Healing of Soft Tissue Wounds

EXCISION OF GINGIVA

Following removal of an excised portion of gingiva (fig. 12-1a), clotting occurs with the formation of a fibrin clot overlying the connective tissue (fig. 12-1b). Within hours, the connective tissue begins to produce granulation tissue (proliferating connective tissue characterized by mitotic activity in the fibroblasts, endothelial cells, and undifferentiated mesenchymal cells), which is soon covered by great numbers of neutrophils both on and within its surface. By this time, there is a base of moderately inflamed connective tissue covered with granulation tissue, a layered zone of neutrophils, and a clot—in that order. Very early in healing, epithelium begins to proliferate from the margins of the wound. Cell by cell, this epithelium migrates *under* the clot and through the neutrophil zone over the granulation tissue (fig. 12-1c).

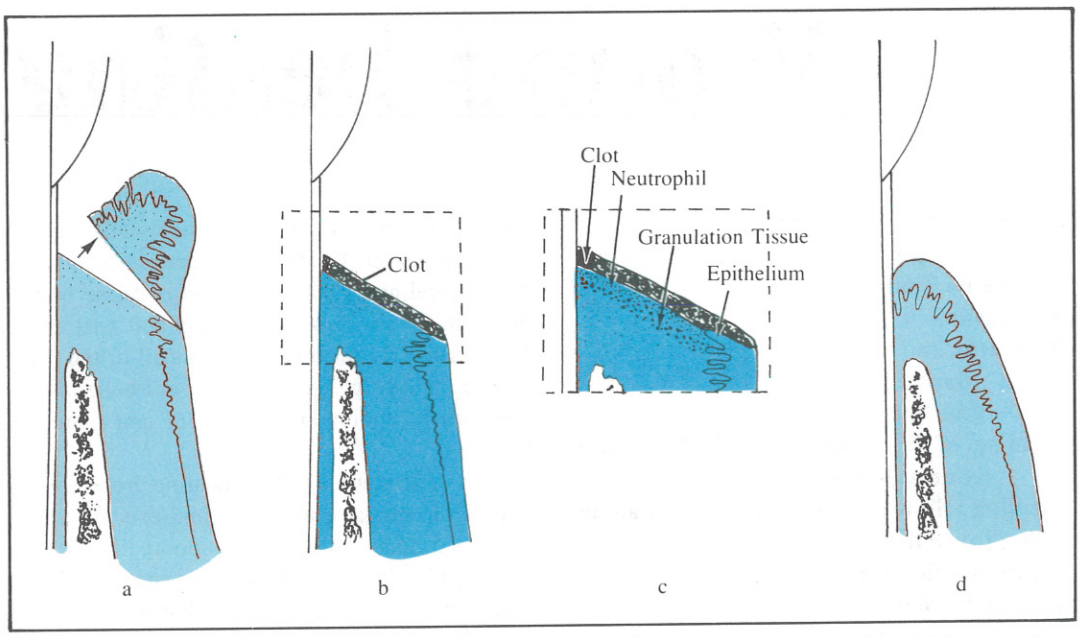

Figure 12-1. Healing after gingivectomy. (a) Excision of gingiva. (b) Formation of fibrin clot. (c) Migration of epithelium through the neutrophil zone between the clot and the granulation tissue. (d) Maturation of formed collagen and formation of new gingival crevice.

Epithelium continues to proliferate until it reaches the surface of the tooth, at which time the epithelial layer is only a few cell-layers thick. While this is occurring, fibroblasts of the granulation tissue produce collagen which, in the early stages of healing, is not completely polymerized and is considered immature. By this time, the clot has been lost, having served its function as a kind of bandage. Proliferation and maturation progress until there is a multilayered covering of epithelium with rete peg formation. A gingival crevice is formed by coronal growth of connective tissue and by apical migration of the epithelial attachment. If healing has progressed relatively free from destructive bacterial agents, the lining of the crevice is composed of a flattened, intact, stratified squamous epithelium.

Maturation of the collagen continues, with the disappearance of granulation tissue, to the point that the formed collagen (the scar) is indistinguishable from the collagen fibers of the attached gingiva (fig. 12-1d). Although clinically the tissue resembles normal gingiva within a few weeks, it will be a number of months before healing is complete and orientation of the fiber bundles occurs.

Even though the surgical procedure has not directly involved bone, there will be some osteoclastic activity on the cortical surface followed by osteoblastic activity. In other words, there is remodeling of bone to some extent, stimulated by surgical trauma to the soft tissue. The remodeling is on a microscopic level and is not of clinical significance.

SIMPLE INCISION

When a sharp instrument cuts through gingiva, processes similar to those just described occur. There are superficial differences based upon the architecture of the wound.

If, following incision, the cut edges are closely approximated, there is only a small space between the wound surfaces to be filled with a clot (fig. 12-2a). This clot serves no known function other than as a "plug" through which granulation tissue grows. Since the elements of the clot are ultimately resorbed, requiring cell effort and time, it is advantageous to have as small a clot as possible in this wound. Healing of this nature by the union of granulating surfaces is termed healing by secondary intention. This is the rationale behind the continued emphasis on wound closure in new attachment procedures throughout the *Syllabus*. The better the wound edge approximation and the smaller the clot, the more rapidly epithelization occurs, thereby sealing off the

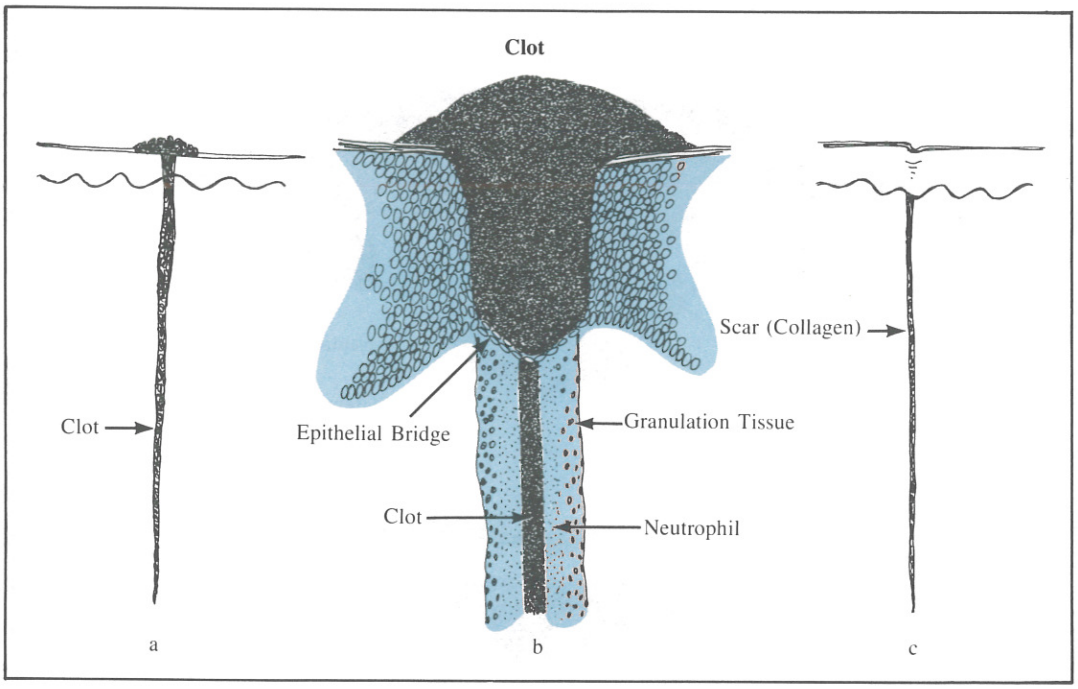

Figure 12-2

slower maturing connective tissues from the oral environment.

Epithelial cells from the wound edges proliferate between the forming granulation tissue and the clot, through the neutrophil zone, as previously described. When these cells join to form a bridge of epithelial cells through the clot (fig. 12-2b), migration apically along the connective tissue ceases. After the epithelial bridge becomes well established, the clot is sloughed off from the epithelial area.

At the same time, clot dissolution is occurring within the depth of the incision as macrophages remove the fibrin and as fibroblasts produce collagen. Once again, collagen maturation occurs with scar formation, which after a few months is clinically indistinguishable from the collagen of the gingiva (fig. 12-2c).

It can be appreciated that if a wound is made in the alveolar mucosa, which lacks appreciable collagen bundles and contains muscle fibers, a clinical scar can remain in evidence for varying periods of time—perhaps indefinitely.

It is also evident that if the wound is a relatively gaping one, the epithelium will migrate farther into its depths along the cut surfaces and, in fact, may cover all exposed maturing granulation tissue and line the wound (fig. 12-3a). In this situation a relatively large mass of clot is sloughed (fig. 12-3b). This is often termed healing by third intention.

Epithelial cells require energy to survive, proliferate, and migrate. Their nutrients are derived by diffusion from the blood vessels. It seems that, in any given wound, the cells can travel only a certain distance from the capillaries, beyond which they lose their source of nutriment. The location of capillaries, then, apparently determines the route of epithelial proliferation.

REATTACHMENT

Reattachment can be defined as the reestablishment of a soft tissue attachment to the tooth after surgical detachment. If gingiva is elevated from a tooth surface, some collagen fibers will tear, leaving fibers embedded in and extending out of the cementum (fig. 12-4a). If the tissue is replaced onto the tooth surface, a clot will be interposed between the fibers in the cementum and the wound surface of the gingiva (fig. 12-4b). Some clinicians believe that if this clot is kept thin, granulation tissue will form, penetrate the clot, and permit the fibers extending from the cementum to become enmeshed with new collagen from the fibroblasts. In some areas cemen-

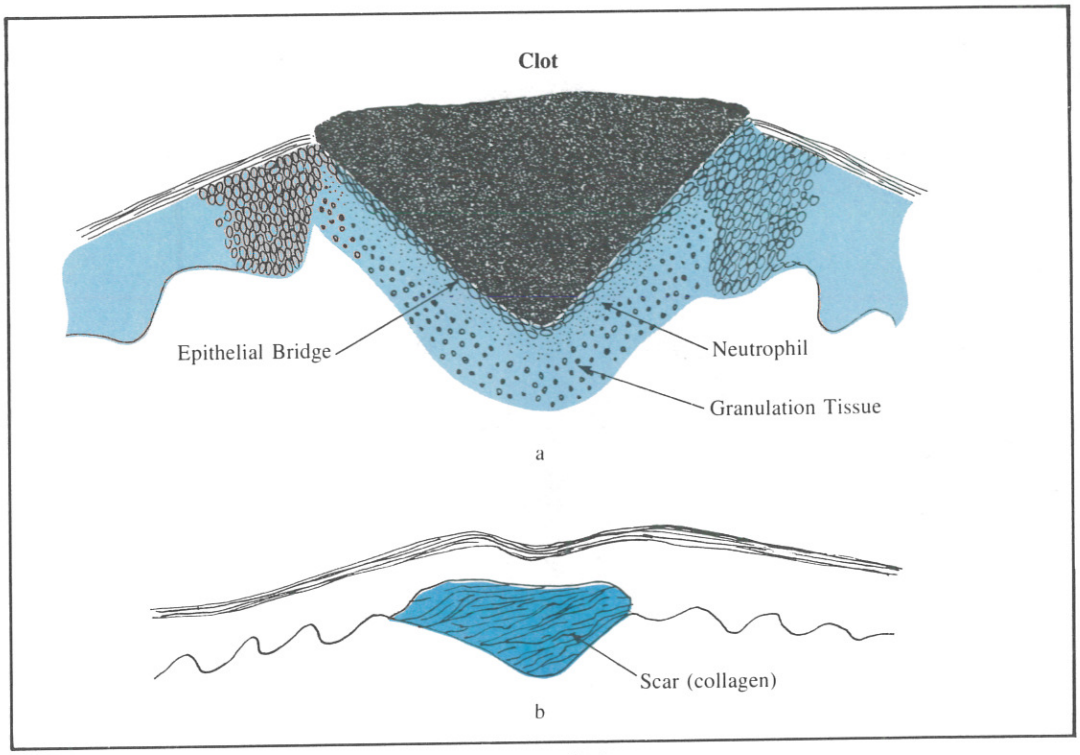

Figure 12-3

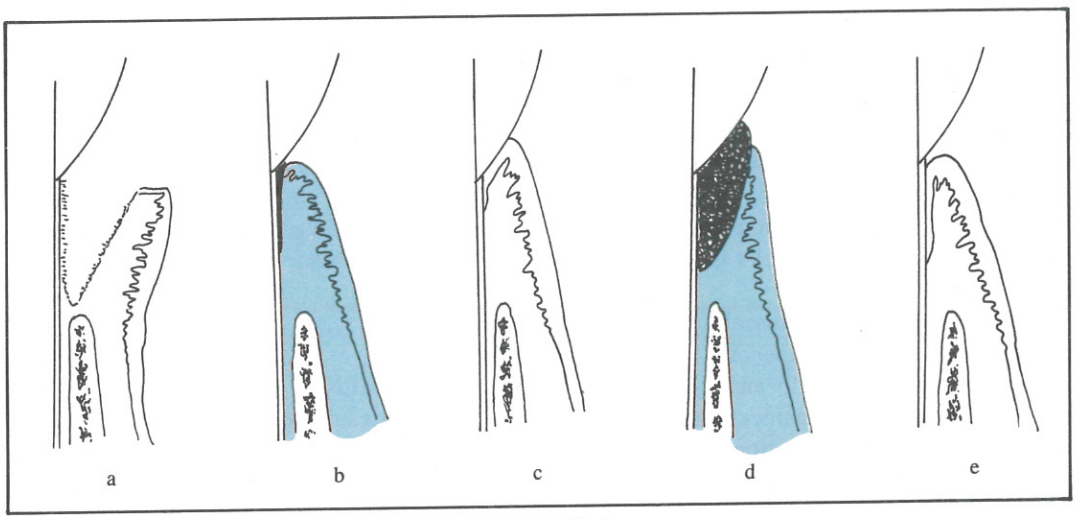

Figure 12-4

tum fibers will be lost, and in these areas new cementum will be laid upon old with the re-formation of Sharpey's fibers. Upon healing, the epithelial attachment may be at its original position on the tooth or it may have moved a few cells apically (fig. 12-4c).

If a large clot is interposed, (fig. 12-4d), epithelium may migrate apically over the wound surface of the gingiva, resulting in an epithelial attachment that is considerably longer and more apically positioned (fig. 12-4e). This occurrence is very unlikely if normal care is exercised in approximating and suturing tissue. Clot thickness can also be controlled by applying gentle pressure for 2 to 3 minutes prior to placement of the periodontal dressing.

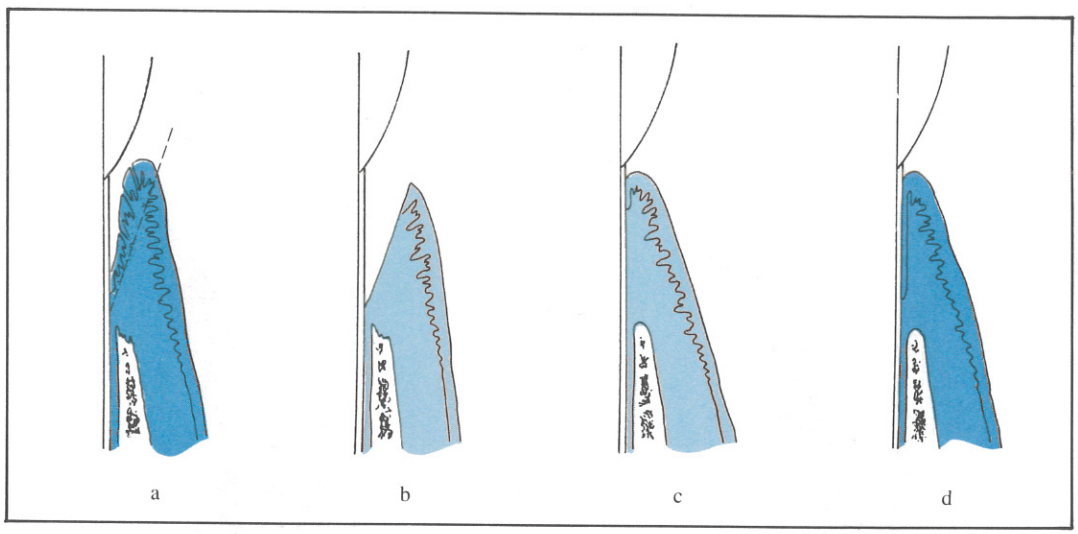

Figure 12-5

NEW ATTACHMENT

New attachment is defined as the establishment of connective tissue to the tooth after surgery at a position more coronal than existed preoperatively. In order to achieve a more coronal attachment of tissue to the tooth, cells capable of producing new cementum and collagen must have access to the tooth surface coronal to the present epithelial attachment. The epithelium lining the pocket prevents such access. This epithelial lining can be removed by scraping the inner wall of the pocket and the epithelial attachment with a sharp curet (subgingival curettage), or by excising the inner aspect of the soft tissue wall of the pocket with a knife (internal bevel incision) (fig. 12-5a). This incision increases access to the cementum of the root (fig. 12-5b), which is often infected by microorganisms within defects of the cementum and within empty spaces formerly filled with Sharpey's fibers. If this cementum is removed by careful root planing and the wound surface is closely readapted to minimize clot size, cellular elements of the granulation tissue will resorb the clot and may produce new cementum, new Sharpey's fibers, and thus establish a new soft tissue attachment (fig. 12-5c).

Of course, it is possible that no new attachment of connective tissue will occur and that a long epithelial attachment will form as shown in figure 12-5d. The result of healing after new attachment procedures is the formation of a new connective tissue attachment at a more coronal level with reestablishment of the gingival fiber apparatus. In either case, the pocket is often eliminated, and the surgical objective is fulfilled.

Confusion has existed for years as to whether new cementum will form on pulpless teeth or teeth with root canal fillings. Recent studies indicate that new attachment can occur on pulpless teeth, teeth left open for drainage, and those with root canal fillings, with the same predictability as for teeth with healthy pulps.

OPEN WOUNDS

At the completion of certain surgical procedures (gingivectomy, gingivoplasty) some areas of the wound will be denuded of epithelium and part of the mucosa. The type of tissue that will be produced during healing depends primarily on the type of tissue composing the wound and its borders. For example, a gingivectomy wound is bordered by gingival tissue and, predictably, gingival tissue will re-form.

Following certain flap operations, areas of bone may be left with only a very thin covering of connective tissue. If gingiva originally covered the bone, then gingiva will re-form. If alveolar mucosa originally covered the bone, alveolar mucosa will usually re-form. Occasionally, and very unpredictably, an intermediate type of tissue may form, clinically resembling gingiva (transitional gingiva). However, the formation of gingiva can be predictably induced in an area of preexistent alveolar mucosa if the

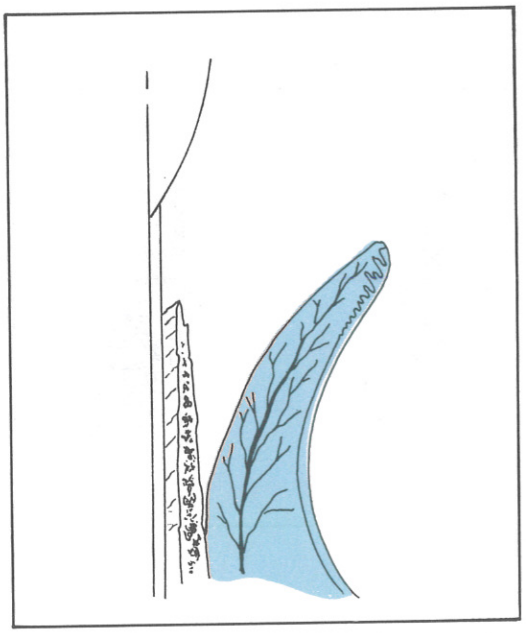

Figure 12-6

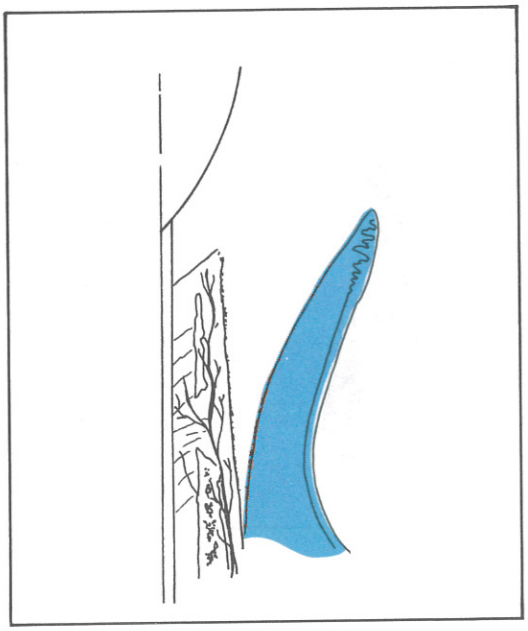

Figure 12-7

apical and lateral borders of the wound are composed of mature gingiva. This gingiva seems, in some way, to stimulate the production of collagen fibers to the exclusion of elastic fibers. This is the rationale for leaving gingival tissue on apically positioned flaps.

HEALING OF FLAPS

A flap is defined as that portion of the gingiva, alveolar mucosa, or periosteum which is elevated or dissected from the alveolar process and which retains a blood supply. Many surgical procedures involve the use of flaps of various designs. There are two methods of raising a flap:

1. Blunt elevation (full-thickness flap) baring the bone surface (fig. 12-6).
2. Sharp dissection (partial-thickness flap) leaving varying amounts of connective tissue over the alveolar process (fig. 12-7).

When flaps are placed back, covering the bone (whether in the original position or at a different site), healing is similar to that of a simple incision in that one connective tissue wound surface is placed against another with an intervening blood clot.

The rationale and technique for the full-thickness and partial-thickness flap will be discussed in chapter 16.

HEALING OF FREE GINGIVAL GRAFTS (FREE SOFT TISSUE AUTOGRAFT)

In contrast to flaps, which retain much of their original blood supply, free grafts are obtained from a donor site, completely freed from their blood supply, and placed at a recipient site. The vasculature at the recipient site then provides all the nourishment for the graft.

The problem of survival of free grafts is one of providing nutrients to the cells of the graft quickly enough to prevent their death in great numbers. For example, the outermost cells of a thick graft often die and are shed during healing. The thin graft (about 1 mm in thickness) stands the greater chance of total survival.

The blood clot at the recipient site should be as thin as possible to permit ready diffusion of nutrients from the recipient site through the clot to the graft. Likewise, the inner surface of the graft should be as smooth as possible to reduce the "pooling" of blood and formation of thicker clots within the surface irregularities.

Since the life of the graft depends on revascularization, the graft must be immobilized at the recipient site.

Free grafts can be successfully established if the operator bases his procedures on an understanding of the healing process.

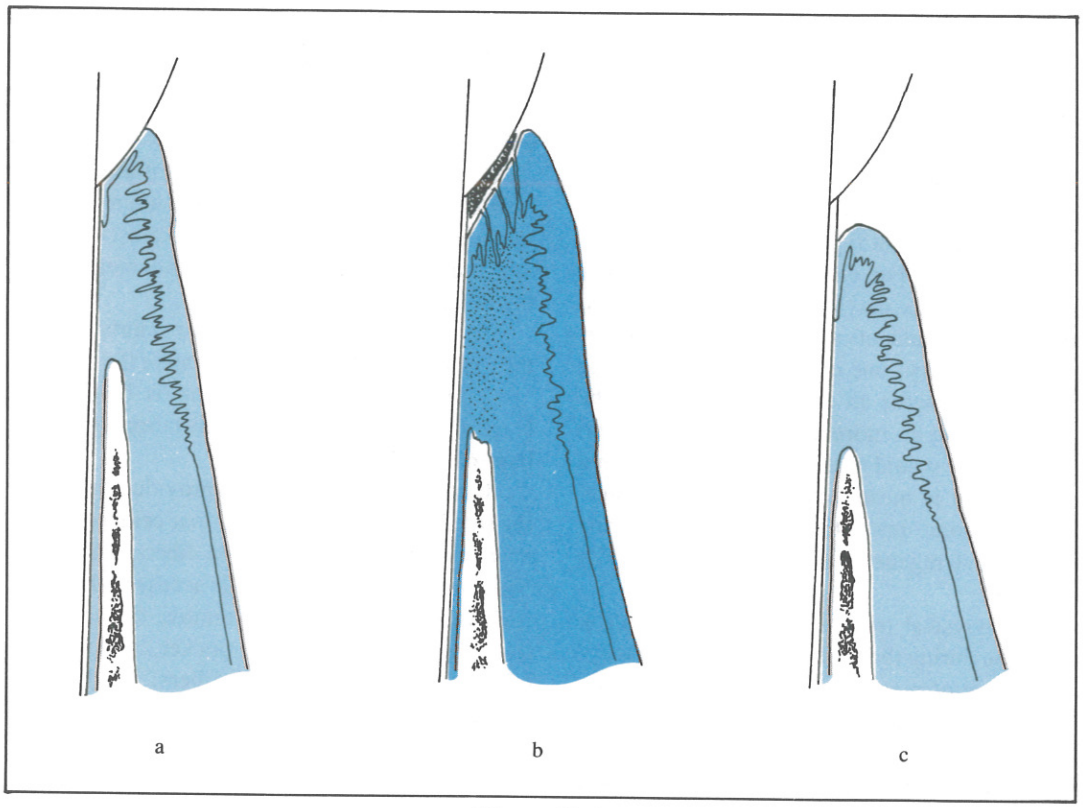

Figure 12-8

HEALING FOLLOWING SCALING AND ROOT PLANING

It has been demonstrated that the removal of plaque and calculus and treatment of exposed root surfaces will result in beneficial changes in the periodontium.

This sequence of events is depicted in figure 12-8. As a healthy periodontium (fig. 12-8a) progresses to pocket formation (fig. 12-8b), chronic inflammation of the gingiva has resulted in depolymerization of some gingival fibers, apical migration of the epithelial attachment, and some loss of crestal bone. The gingiva itself is somewhat enlarged as a result of edema. Young vascular connective tissue is present within the gingiva, as are many lymphocytes, plasmocytes, and neutrophils—all the elements of defense and repair.

These defensive elements have little success against the colonized bacteria, and the granulation tissue has little chance to effect repair because of continued tissue damage from products of the microorganisms. This is the condition commonly found in these lesions—ineffectual defense and frustrated attempts at repair.

If assistance is provided in the form of scaling and root planing, and the establishment of effective plaque control procedures, inflammation will be resolved. The edema will subside and the granulation tissue can proceed to repair with regeneration of gingival fibers. As maturation progresses free from the effects of plaque, further shrinkage will occur until a condition similar to that in figure 12-8c is reached.

Healing Involving Bone

GENERAL PRINCIPLES

Some important principles concerning bone physiology are in order. Bone undergoes remodeling throughout life. In health, osteoblastic activity balances osteoclastic activity. This balance

can be disturbed by changes in functional demands on the bone, by disease, or through other causes. Furthermore, any surgical procedure affects bone to some extent. The severity of the change depends on several factors, including the type of bone (cortical or cancellous), the thickness of the bone, what is done to it during the procedure, and what type of covering it has following surgery. For example, if thin radicular bone overlying a maxillary cuspid were bared during surgery and left without soft tissue coverage after surgery, one could expect that during the healing process all denuded bone would be either resorbed or, more likely, sequestrated and that little of it would regenerate. If interproximal bone, which is mostly cancellous, was treated similarly, some resorption would occur during healing, but regeneration would be practically complete.

Any physical trauma to bone will result in resorption during the healing process, with varying degrees of regeneration during maturation.

When performing periodontal surgery, one should remember that:

1. Bone should be covered by soft tissue after surgery, if at all possible.

2. The thicker the soft tissue covering over bone during and after the procedure, the less will bone be affected by surgery.

3. Thin bone can be permanently lost more easily than thick bone. Thick bone, as seen interproximally, has sufficient intra-alveolar blood supply (cancellous bone) to withstand the loss of supply from the supraperiosteal vessels as a result of flap surgery. Conversely, thin radicular bone is almost totally dependent on the blood supply from the supraperiosteal vessels (lacks cancellous bone) and can be readily lost if this supply is compromised. All cuspids, first bicuspids, and the mesial roots of maxillary first molars should be considered to have this thin bone. (These teeth may even have bony dehiscences or fenestrations, as discussed in chapter 1, "The Periodontium.") For that matter, the radicular bone over all facial root surfaces should be considered carefully in the design and execution of surgical procedures.

INFRABONY DEFECTS

Many procedures are performed to stimulate new bone formation to repair an osseous defect. Figure 12-9a represents a mesiodistal section through the proximal surfaces of two teeth and the interproximal bone and gingiva. A chronic infrabony defect is diagramed with the intent of illustrating three conditions common to practically all such chronic lesions:

1. Transseptal fibers are present as discrete bundles extending from the cementum of one tooth, sweeping obliquely over the crest of bone and its defect, and continuing to the cementum of the adjacent tooth.

2. A cortical surface has been maintained on the bony surface that forms the wall of the defect.

3. The epithelial attachment is below the crest of the bone at a certain distance from the base of the defect.

If new bone is to form and provide support to the tooth, it will be necessary that cells produce new cementum, new collagen fibers, and new bone. In order to give the cells access to the area involved, it is necessary to remove the connective tissue portion of the pocket, the pocket epithelium and transseptal fibers. As this is done, granulomatous tissue within the defect will also be removed.

After the soft tissue has been removed, the cortical bone lining the defect should be perforated (intramarrow penetration) to permit quick egress of granulation tissue that contains pluripotential cells for re-formation of the attachment apparatus (fig. 12-9b). Likewise, complete

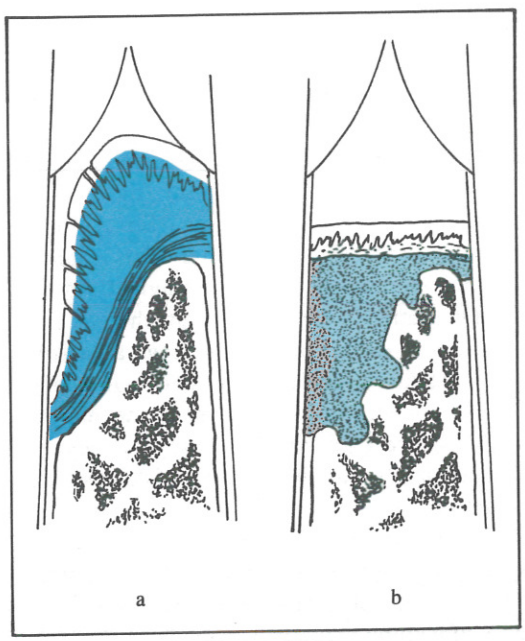

Figure 12-9

wound closure is a prerequisite for successful healing.

Ideally, complete restoration of cementum, bone, and periodontal ligament will occur. Occasionally, however, only partial regeneration will result. The predictability of success for such new attachment procedures is discussed in chapter 18, "Management of osseous defects: grafting."

OSSEOUS AUTOGRAFTS

Autogenous bone grafts have been used quite successfully for many years in the management of osseous defects of the periodontium. However, the precise mechanism through which these grafts contribute to healing and regeneration remains unclear.

It has been postulated that the primary effect of autogenous grafts is osteogenic stimulation. Autogenous *marrow* grafts, however, are believed to act not only as osteogenic stimulators but as actual bone producers, because of their high content of cells with osteogenic potential.

Another possible benefit of grafts is that they act as a mechanical block to the downgrowth of epithelium into the wound. They are thought also to act as trellises for the proliferation and migration of cells and vessels within the wound. Generally, cancellous autogenous bone has produced better clinical results than cortical bone. This may be owing to the remaining vitality of cells within the marrow spaces as well as to the trellis effect.

Fate of Autografts

The fate of the calcified portions of autografts (and of other materials used) has been documented as follows:

1. The graft may retain vital osteocytes and be built upon by new bone. Such osteocytic life will be maintained only when the cell is close enough to a nutrient source (Ham* suggests "a fraction of a millimeter"). This suggests that osteocytes within a comparatively large fragment will die.

2. The graft may be necrotic (no vital osteocytes) but be built upon by new bone and later replaced by new bone.

3. The graft may remain as an inert, nonvital fragment, playing no apparent role in the healing process.

4. The graft may be completely resorbed during healing, having played no apparent role.

5. The graft may be necrotic, play no apparent role, and be exfoliated during healing. Exfoliation may occur months after the surgical procedure.

Other Considerations in Wound Healing

NUTRITION AND SYSTEMIC DISORDERS

Healing occurs on a cellular-molecular level requiring the expenditure of energy and the utilization of nutrients as required for anabolic and catabolic activities. Any disorder which interferes with ingestion, digestion, absorption, or effective transport and utilization of foods will interfere with healing. Such disorders as diabetes, deficiencies in vitamins and foods (especially protein), and severe hormonal imbalances can be expected to retard healing.

AGE

Age by itself seems to have no bearing on healing following periodontal surgery. General health seems to be of paramount importance.

ASEPSIS

Asepsis is a basic requirement for success in all surgery performed in the oral cavity. Periodontal surgery must be performed in a manner that will prevent introduction of foreign pathogens into the surgical field which could result in infection and delayed healing. Hat, mask, and sterile gloves, instruments, and drapes should be routine in the performance of all surgical procedures.

HEALING RATE

It seems that there is a maximum speed at which various cells can clean up cellular debris, produce materials, move through tissue, or perform

*Ham, Arthur W. Histology, ed. 5. Philadelphia, Lippincott, 1957, p. 385.

any number of different tasks. To date, there is no way to hurry them along. Perhaps the best one can do is to avoid slowing them down.

Wound Healing Applied to Periodontal Surgery

A review of this chapter will serve as a reminder that there are many principles of wound healing which apply directly to success or failure in periodontal surgery. The following is a summary of the more important principles that have been discussed:

1. A gingival wound may appear normal within a few weeks; however, it will be a number of months before healing is complete and fiber bundles have formed.

2. The better the wound edges approximate and the smaller the clot, the more rapidly epithelization occurs, thereby sealing off the slower maturing connective tissues from the oral environment.

3. In flap procedures, clot thickness should be kept at a minimum between tooth and wound surface in order to permit either *reattachment* of connective tissue fibers at their original level or *new* attachment at a more coronal level.

4. For predictable maturation of gingival tissue in apically positioned flaps, gingival tissue should be maintained on the margin of the flap.

5. *Thin* free grafts placed on a recipient site over a *thin* blood clot afford an excellent opportunity for graft survival.

6. Plaque, calculus, and infected cementum *must* be removed for successful wound healing.

7. Bone should be covered by soft tissue when surgery is finished.

8. The thicker the soft tissue covering over bone during and after the procedure, the less the bone will be affected by surgery.

9. Thin bone can be permanently lost more easily than thick bone.

10. In the management of infrabony defects it is necessary to remove *all* the connective tissue portion of the pocket (epithelium, transseptal fibers, granulomatous tissue). Also, the cortical bone lining the defect should be perforated to permit egress of pluripotential cells.

13 Principles of periodontal surgery

- Reasons for surgery
  - Providing access
  - Restoration of missing parts of the periodontium
  - Restoration of bony architecture
  - Restoration of attached gingiva
  - Indications for surgery
- Presurgical considerations
  - Prescaling
  - Full mouth surgery
  - Preoperative medication
    - Premedication
    - Antibiotics
    - Anesthesia
- Surgical considerations
  - Planning the surgery
  - Instrumentation and flap design
  - Wound closure
  - Control of bleeding
  - Suturing
  - Periodontal dressings
    - Function
    - Eugenol dressing
    - Noneugenol dressing
    - Placement
    - Removal
- Postsurgical considerations
  - Postoperative instructions
  - Postoperative problems
- Limitations of surgery

With the exception of abscess treatment, the performance of periodontal surgery in the absence of plaque control or with no assurance of effective plaque control is a waste of time.

If we can ever fortify the periodontium against plaque, perhaps we can actually cure periodontal disease instead of merely arresting it. Surgery does not increase resistance, nor does it reduce bacterial growth. Consequently, if plaque formation continues postoperatively, periodontal disease will recur.

Reasons for Surgery

PROVIDING ACCESS

The patient who earnestly tries to control plaque but whose efforts are frustrated by uncleansable pockets will benefit from increased access to tooth surfaces. Surgery can provide this access. Surgery will also permit the dental officer to visualize and treat the root surfaces and osseous defects.

RESTORATION OF MISSING PARTS OF THE PERIODONTIUM

In addition to providing access, surgery can restore missing parts of the periodontium, specifically, bone and attached gingiva.

RESTORATION OF BONY ARCHITECTURE

Bony defects are undesirable for two reasons. First, since the epithelial attachment tends to follow the contour of the defect, access to the root surface for plaque control is virtually impossible in infrabony pockets. For example, the position and contour of the gingiva in figure 13-1a, and the bony configurations in figures 13-1b and 1c, prevent cleansing of the pocket

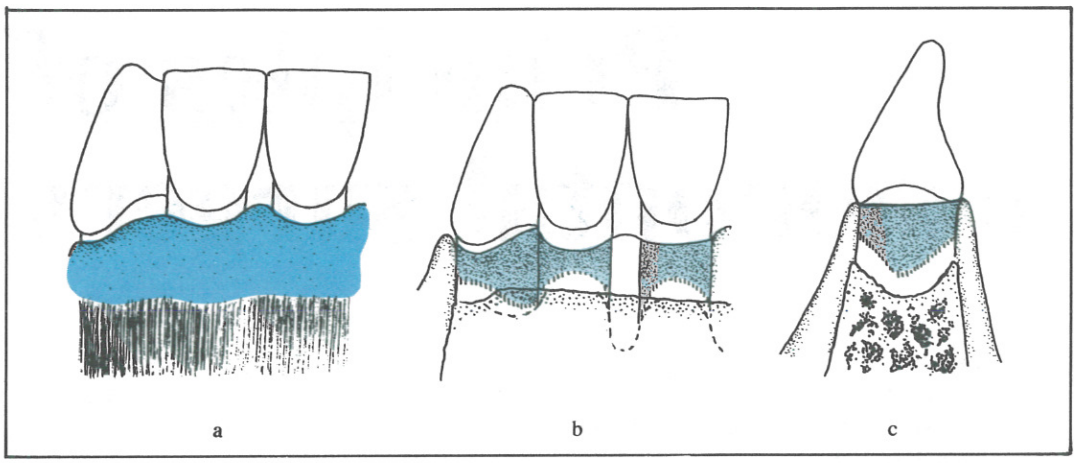

Figure 13-1

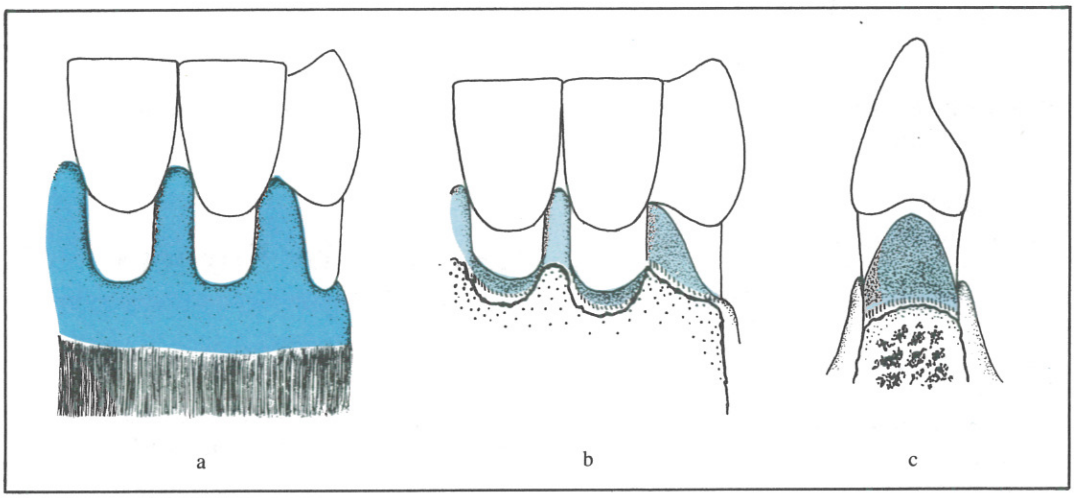

Figure 13-2

areas shown by shading. In contrast, the gingival contour in figure 13-2a and osseous contours in figures 13-2b and 2c permit access to the shaded areas. Second, bone loss decreases support to the tooth. Although it is difficult to increase the amount of bone support, it is often possible to eliminate the defect, facilitate plaque control, and thus prevent further bone loss. The treatment of bony defects will be discussed in subsequent chapters.

RESTORATION OF ATTACHED GINGIVA

Some width of attached gingiva is necessary for optimal health. Sometimes there will be an inadequate amount, or even none, present. The problem is to ensure an adequate zone of attached gingiva following healing. This can be accomplished surgically by grafting, that is, by preparing the involved area for reception of keratinized gingiva obtained from another area. Another approach is to remove part of the tissue at the involved area and to permit its replacement by attached gingiva or transitional gingiva, as described in chapter 12, "Wound healing."

In short, keratinized gingiva can often be grafted or grown at a desired site. This can also be done as a preventive measure; for example, where there is barely adequate attached gingiva on a tooth requiring a full crown. Following crown preparation, impression-making, and a

period of temporary coverage, this minimal zone of attached gingiva may become permanently detached. The problem will not arise if a broader zone is established *before* prosthodontic treatment begins.

INDICATIONS FOR SURGERY

The decision to operate should be based on the patient's effectiveness in plaque control and on periodontal architecture after initial treatment. It should not be based solely on pocket depth.

If surgery is required, the patient should be made aware of it early in treatment and should understand its role in the overall treatment plan. The highly motivated, interested patient will accept, almost eagerly at times, the necessity for surgery when he realizes that his treatment would be incomplete without it. Unfortunately, some patients have the false idea that surgery in itself is curative: "I had a gingivectomy 3 years ago, and I still have problems!" This fallacy should be dispelled early in treatment.

When the time for surgery arrives, it will be helpful for the patient to understand the general objectives of the procedure, but without being burdened with explanations of specific techniques. He should be informed in advance that he will have to wear a dressing for several days.

Presurgical Considerations

PRESCALING

Controversy still exists about the desirability of scaling and root planing before surgery. Some clinicians oppose this procedure for the following reasons:

1. It wastes time, causes needless discomfort, and can be accomplished more easily during surgery.
2. Preoperative scaling and root planing subjects the patient to additional bacteremia and its possible sequelae.
3. With proper lavage and aspiration, bleeding during surgery is not really a problem.
4. Healing time is not affected.
5. The competent practitioner should be able to tell in advance whether surgery is needed.

Most practitioners, however, advocate scaling and root planing before surgery for the following reasons:

1. It permits immediate improvement in plaque control.
2. It permits fair evaluation of the patient's willingness and ability to control plaque by providing clean, smooth root surfaces.
3. It provides time for evaluation of the patient's healing capacity. Even the most competent clinician cannot always anticipate the extent of healing that will occur after debridement of the tooth.
4. After debridement, tissue architecture may change drastically, suggesting surgical procedures different from those contemplated originally.
5. Double bacteremias are not significant except in certain circumstances (e.g., rheumatic heart disease and cardiovascular prosthesis implants).
6. Bleeding during surgery may be excessive if scaling and root planing are not done preoperatively.
7. Improved tissue tone and consistency enhance resection techniques, facilitate the elevation of partial-thickness flaps, and improve the chances for primary wound closure.

FULL MOUTH SURGERY

Successful periodontal surgery demands full attention to detail in handling both hard and soft tissue. This exactness may require longer appointments and be fatiguing to both patient and operator. Unless the full mouth operation involves few teeth, it is better to perform surgery in quadrants or sextants. This will be determined by the type and location of the surgery required.

If circumstances force the dental officer to accomplish all surgery at one appointment, it may be prudent to seek help from additional operators for surgery in shifts. This approach is rarely necessary.

PREOPERATIVE MEDICATION

Premedication

Perhaps the best preoperative sedative for the patient is an appreciation for surgery, combined with confidence in his dental officer. Should premedication for apprehension be needed, 25 mg of meperidine hydrochloride and 25 mg of

promethazine hydrochloride given orally 30 to 45 minutes before anesthesia provide sedative, amnesic, antiemetic, antihistaminic, and analgesic actions. Patients premedicated with a sedative or an amnesic should always be accompanied by a responsible adult and should never be permitted to drive a vehicle on the day of surgery.

Antibiotics

There are strong advocates for and against the routine use of antibiotics for prophylaxis in periodontal surgery. There is little evidence that antibiotics lower the incidence of infection or improve wound healing. There is strong evidence, however, that antibiotics will suppress plaque formation. Bacterial colonization during the initial stages of healing in new attachment procedures can result in surgical failure. For this reason, antibiotics are recommended for plaque suppression during bone grafting procedures. Broad spectrum antibiotics (tetracycline) 250 mg q.i.d., are prescribed on the day of surgery and for 7 days thereafter. It should be reemphasized that antibiotics are always prescribed for patients who require prophylaxis for such disorders as congenital heart disease or valvular damage. Tetracycline should not be taken with food because of the likelihood of impaired absorption. Also, tetracycline may cause discoloration of developing teeth, and caution should be observed in prescribing it for women in the third trimester of pregnancy, or for children with developing dentitions.

Anesthesia

Periodontal surgery is normally done under local anesthesia. All local anesthetic agents are toxic drugs; and the clinician should be aware that the method of injection, the vascularity of the injected tissue, and also the dosage have an effect on the patient. He should know the safe dose range of the anesthetic he has selected. For example, the dose range of lidocaine hydrochloride, as listed in the United States Pharmacopeia* is up to 500 mg, or 25 ml of a 2% solution. Monheim† suggests the maximum dosage for the ambulatory dental patient is approximately 300 mg (or 15 ml of a 2% solution of lidocaine). In short, a clinician can use 8 cartridges (14.4 ml total) of lidocaine without a vasoconstrictor before he approaches the maximum limit as suggested by Monheim.

Most anesthetics used in dentistry contain epinephrine. The New York Heart Association‡ has recommended that for any one session, no more than 0.2 mg/ml of epinephrine be used. It will take 20 ml (about 10 cartridges) of a local anesthetic with 1:100,000 epinephrine to reach the recommended level. If a local anesthetic with 1:50,000 epinephrine is used, the level will be reached after 10 ml (approximately 5 cartridges). Rarely is it necessary to use 5 anesthetic cartridges for periodontal surgery unless full mouth surgery is performed. In this instance, it may be safer to use an anesthetic with 1:100,000 epinephrine.

Anesthetics with 1:50,000 epinephrine are generally preferred in periodontal surgery, because this concentration of epinephrine appears to result in less bleeding at the operative site, a factor which improves visibility.

Injection of any local anesthetic in dentistry should be accomplished with aspirating syringes, and at a rate which does not exceed 1 ml/min.

Surgical Considerations

The following general observations concerning surgery should be helpful.

PLANNING THE SURGERY

Flexibility of mind and imagination are required in periodontal surgery. When the mandibular right quadrant, for example, is to be treated surgically, each area should be examined to determine what problems exist and what needs to be

*Pharmacopeia of the United States of America, United States Pharmacopeia, 12601 Twinbrook Parkway, Rockville, Md. 20852.

†Monheim, L. M. Local Anesthesia and Pain Control in Dental Practice, ed. 4. Saint Louis, The C. V. Mosby Co., 1969, p. 145.

‡Special Committee of the New York Heart Association, Inc., J. Am. Dent. Assoc. 50:108, 1954.

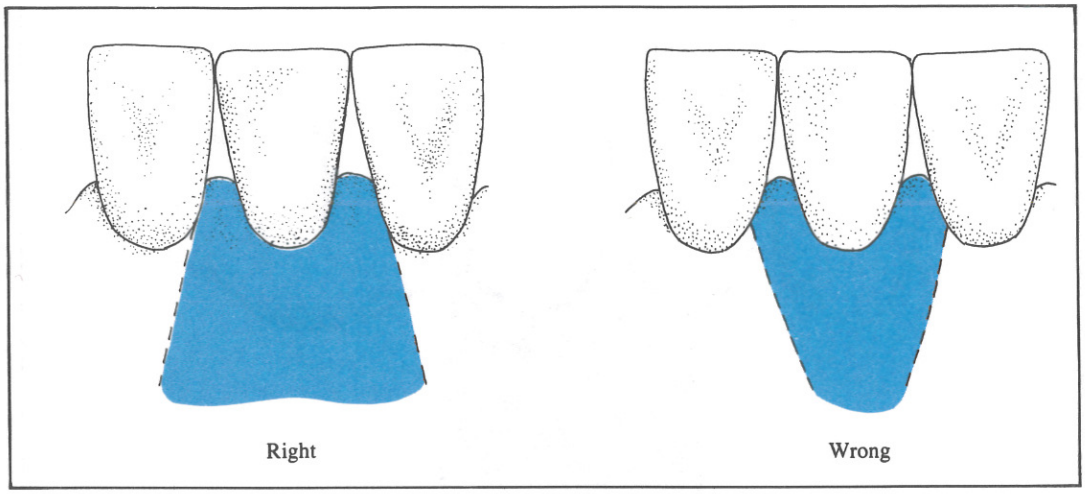

Figure 13-3

done to solve them. On the second molar, there may be a pocket that extends below the mucogingival junction on the facial side. On the bicuspids, there may be an inadequate zone of attached gingiva on the facial aspect and shallow pocket formation on the lingual aspect. In this quadrant, a gingivectomy may suffice on the lingual side; whereas the facial side will require a flap approach in the molar area and a gingival graft in the bicuspid area.

INSTRUMENTATION AND FLAP DESIGN

Cutting and planing instruments must be sharp. Dull instruments macerate tissue, complicate healing, and frustrate the operator. If dull instruments are found in a set, another set should be opened. Extra Bard-Parker blades should be readily available.

Continuous awareness of the location of the cutting edge of a knife will prevent conversion of a flap into a free graft.

Cells die when abused. This basic fact suggests a healthy respect for tissue during its manipulation. When a flap is being retracted after elevation, for example, there will be less trauma if the retractor is held gently but firmly against bone instead of against the undersurface of the flap. If retraction is needed on the palate, a suture passed through the flap and tied to a tooth on the other side will suffice.

So-called vertical relaxing incisions or access incisions extending toward the palatal vault or along the lingual alveolar plate in the mandible should be avoided. Incisions of this type, particularly in the palate, can interfere with the blood supply to tissue mesial to the incision. These incisions are also difficult to suture and often heal slowly with noticeable discomfort to the patient, especially those incisions in the mandible. These problems can usually be avoided by extending the initial flap incision to include a few teeth mesial or distal to the area of instrumentation. If vertical incisions are utilized on the facial side, they should be designed so as not to compromise the blood supply of the flap (fig 13-3). Also, vertical incisions should be made at line angles to preserve the interdental papillae for suturing and to prevent necrosis of the wound edge; and under no circumstance should vertical incisions be made over the midfacial (radicular) surface of roots (fig. 13-4).

If a periodontally weakened tooth is used as a fulcrum for elevation of a flap (especially a palatal flap), accidental removal of a tooth can result.

Bone contouring can be done with sharp chisels or rotating instruments. Caution should be exercised, especially if hand pressure is used, to prevent slipping of the instruments. If a handpiece and burs or stones are used, a saline coolant should bathe the area. Ultraspeed cutting should be done with the lightest pressure and intermittent contact. Continuous rinsing during ultraspeed cutting is not very effective, because the

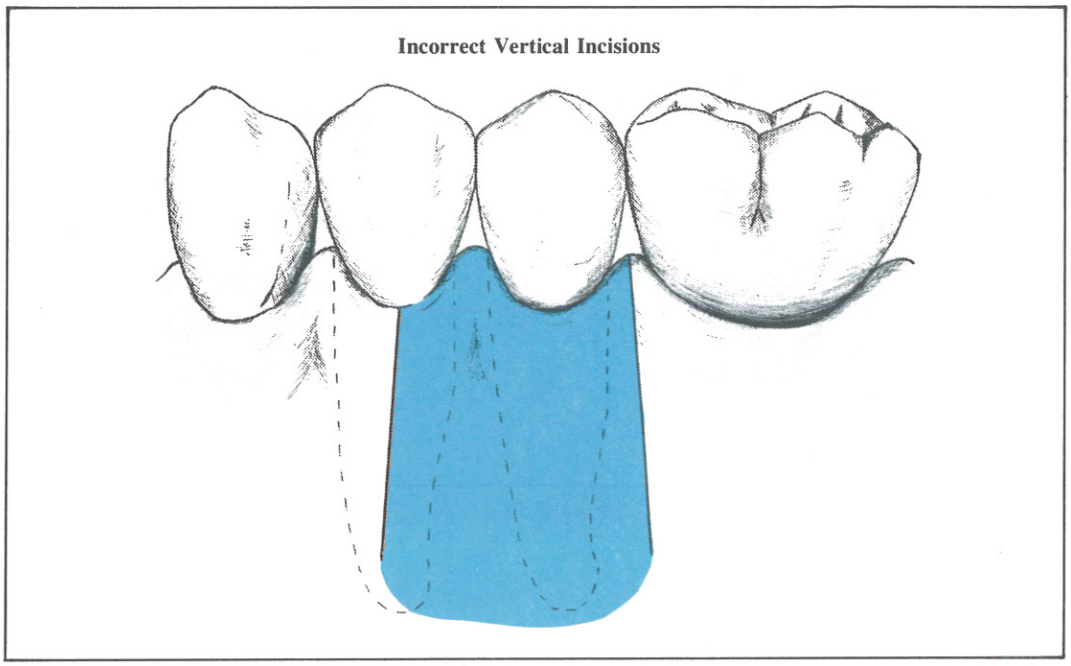

Figure 13-4

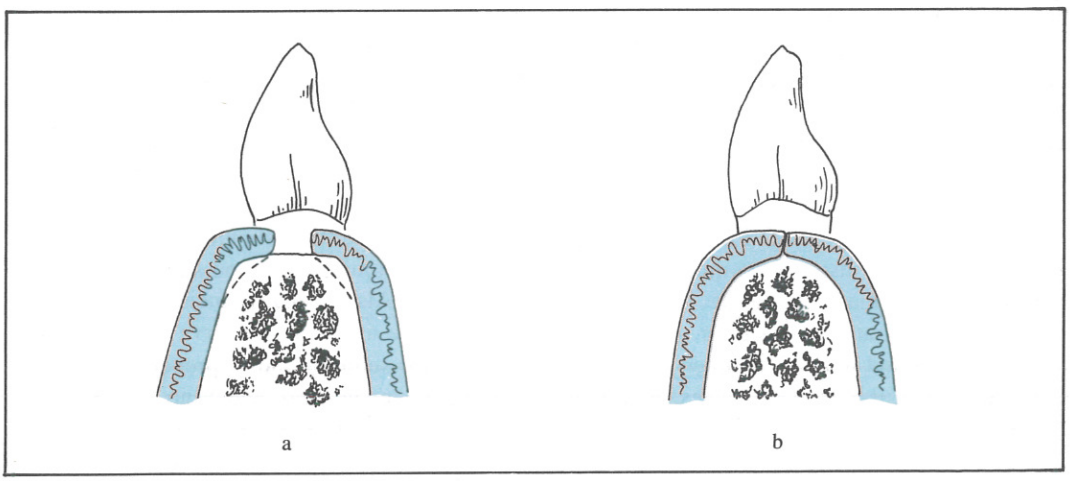

Figure 13-5

vortex of air around the bur or stone prevents the coolant from contacting either bone or bur. The bone may therefore become dry unless it is rinsed intermittently.

WOUND CLOSURE

As clinical experience is gained in new attachment procedures, it becomes increasingly apparent that primary closure to seal off the oral environment plays a major role in success or failure. Consequently, techniques are being devised which permit the greatest opportunity for primary closure in the interproximal region. The following suggestions are offered:

1. If necessary, perform interproximal osteoplasty to improve approximation of wound edges (figs. 13-5a and 5b).

2. Maintain as much of the interdental papillae as possible by utilizing a scalloped incision (fig. 13-6).

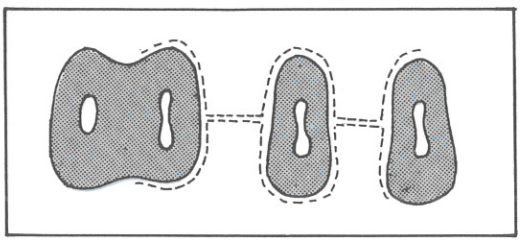

Figure 13-6

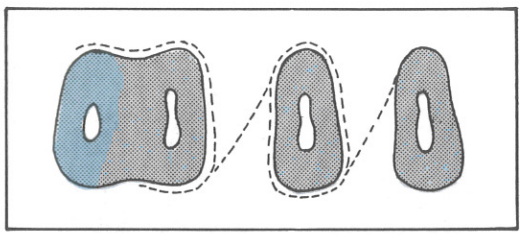

Figure 13-7

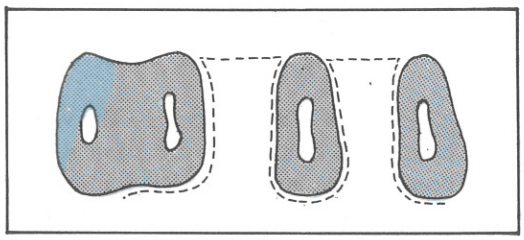

Figure 13-8

3. When possible, make a diagonal incision to increase the length of interproximal wound surface for suturing (fig. 13-7).

4. Another method of achieving primary wound closure is to incise the papillae on the facial or the lingual side (fig. 13-8) and retract it through the interproximal space. On completion of surgery, return the tissue through the interproximal space and suture it.

As yet, no good technique is known to the authors to obtain adequate wound closure in furcation areas.

CONTROL OF BLEEDING

Should a bleeding problem arise, pressure should be applied directly to the bleeding site. Bleeding from the attached base of a flap can often be stopped by replacing the flap and applying pressure over the area. The pressure should be heavy enough to overcome capillary or arteriolar pressure but not heavy enough to cause tissue damage. Occasionally, bleeding occurs interproximally when a flap has been elevated. This will often stop as soon as the granulomatous tissue has been removed. If a vessel bleeds out through a nutrient canal of bone, cotton pellets soaked in saline solution can be packed interproximally. This often provides quick hemostasis. If cotton pellets and pressure do not control a bone-bleeder, pressure applied with a metal instrument can close the canal to stop the bleeding.

Following tissue resection, powdered thrombin can be applied topically at persistent bleeding sites on raw surfaces, particularly where capillary oozing may not be controlled by pressure alone. Thrombin is normally supplied in bottles of 5,000 N.I.H. units and is quite expensive. Since each bottle can be used only on a one-time basis, thrombin should be used only when necessary and not simply as a convenience to the operator. THROMBIN MUST NOT BE INJECTED INTRAVENOUSLY or otherwise allowed to enter large blood vessels, since extensive intravascular clotting and even death may result.

Before suturing and dressing, the wound site should be inspected for loose debris, loose blood clots, calculus on roots, and tissue tags. If possible, small tissue tags should be removed with scissors, forceps, or curets, for these often persist as bleeding sites, become necrotic, and complicate healing.

Bleeding should be stopped before the dressings are placed. The hemostatic effect of dressings is not great, nor are they adequate pressure dressings. Remember to strive for minimal blood clots in new attachment procedures. This is accomplished by applying gentle pressure to the flap or graft for 2 to 3 minutes with gauze soaked in saline solution prior to the placement of the dressing.

SUTURING

The extensive use of flaps and grafts in periodontal surgery has concomitantly increased the need for suturing. The following guidelines may help when deciding the type of suture material and technique to use.

1. The smallest and least reactive suture material compatible with the surgical procedure

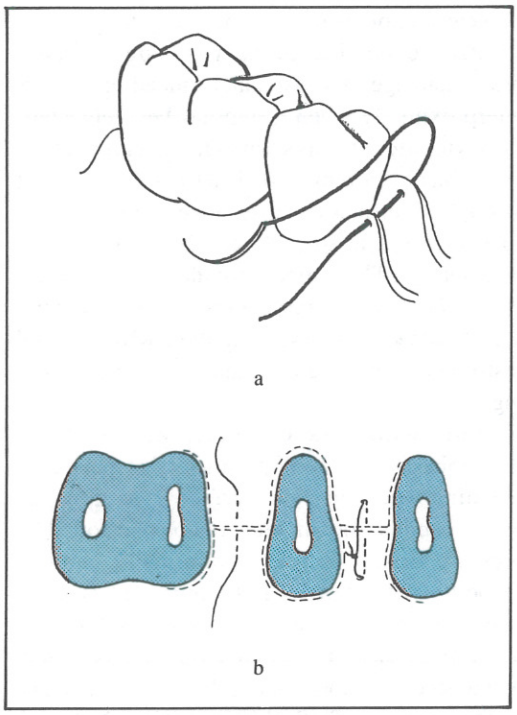

Figure 13-9

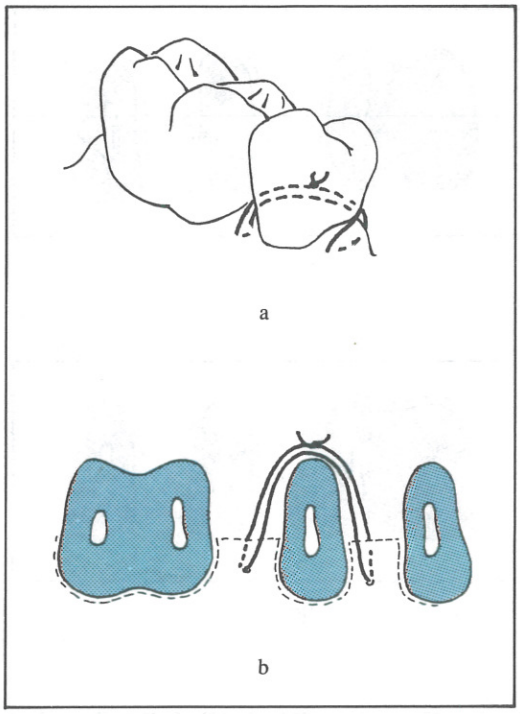

Figure 13-10

should be used—monofilament sutures produce a milder tissue reaction than silk and are often advocated for this reason.

2. A circle cutting, swaged needle is the least traumatic of needles and is likewise recommended for use in periodontal surgery.

3. There are any number of good techniques of suturing described in the periodontal literature. The three types of sutures used most frequently are the interrupted, the sling, and the vertical mattress.

a. *Interrupted Suture*. This suture can be used for virtually all flap and graft surgery. It has its greatest application when both tissue margins require the same amount of tension, as in interproximal tissue approximation (figs. 13-9a and 9b).

b. *Sling Suture*. The sling suture encircles the tooth and is used primarily when a flap has been raised on one side of the tooth and it is undesirable to tie to the opposite side. Sling sutures are often used as suspensory sutures to hold a flap coronally, such as the laterally positioned flap or the double papillae flap (fig. 13-10).

c. *Vertical Mattress Suture*. The vertical mattress suture offers the advantage of keeping the suture material from under the margin of the flap. This suture is often used for interproximal tissue approximation over bone grafts and in excisional new attachment procedures and replaced flaps (fig. 13-11).

Precise suturing is extremely important in periodontal surgery. When performed properly, suturing will stabilize flaps in the desired position, ensure primary wound closure, control bleeding, and help reduce postoperative pain.

PERIODONTAL DRESSINGS

Function

The most important function of periodontal dressings is to protect the underlying wound from trauma during mastication or while plaque control devices are in use. When the surgeon feels that the tissue has healed enough to allow resumption of plaque control procedures in the surgical area, the dressing will have served its purpose and need not be replaced. The dressing also has an obtundent effect and, to some extent, controls pain.

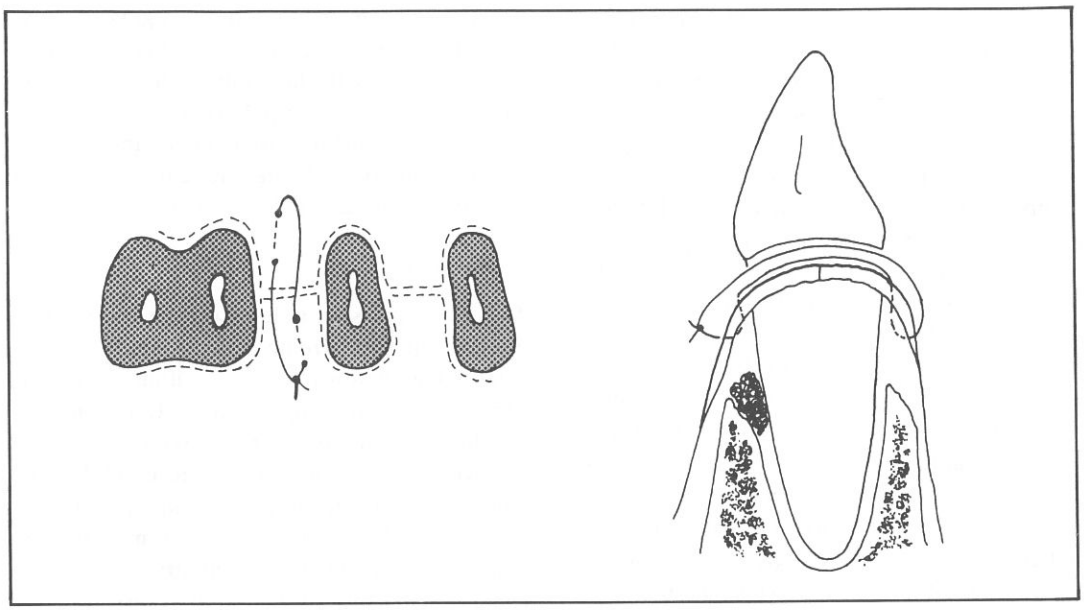

Figure 13-11

There are two principal types of periodontal dressings in use: the eugenol and the noneugenol.

Eugenol Dressing

The powder portion of a standard dressing (PPC) is composed of zinc oxide and a combination of the following dry ingredients: kaolin, powdered asbestos, rosin, tannic acid, and zinc acetate. The liquid portion is eugenol, with or without thymol, and cottonseed oil.

Sterile gloves, mixing pad, and tongue depressor are used to prepare the dressing.

The powder is added to the liquid in small portions until the mix thickens. The "dough" is then kneaded with the fingers, and more powder is added to obtain a thick, puttylike material. It is then rolled into a small round strip about ¼-inch in diameter and cut in pieces 1 to 2 inches long. The strips are powdered, wrapped in foil, and placed in a freezer. Frozen dressings may be stored several months.

Noneugenol Dressing

Noneugenol dressings gained popularity when surgical techniques that left bone exposed were in use. Eugenol was implicated as an irritant to osseous tissues, which stimulated the development of noneugenol dressings. Bone is now rarely left exposed, but in current techniques, there are still many clinicians who prefer this type of dressing.

Baer-Sumner Dressing. The noneugenol Baer-Sumner dressing contains zinc bacitracin. Its main advantage is its ability to suppress odor-forming bacteria which usually develop when dressings are worn. The Baer-Sumner dressing is not as rigid as a eugenol dressing. For this reason, it is often used as a preliminary dressing and then covered by a standard eugenol dressing. It consists of a powder and a base in the following proportions:

Powder (to make 1 gram):

Zinc oxide	0.43 Gm
Rosin powder	0.57 Gm
Zinc bacitracin*	3,000 units

Base:

Zinc oxide	21 Gm
Hydrogenated vegetable fat	39 Gm

Base and powder are mixed on a waxed pad with a tongue blade or heavy spatula until the mix becomes crumbly. Wearing sterile rubber gloves, the mixer continues to knead more powder into the base until a doughlike consistency is reached.

*Some clinicians prefer to substitute neomycin for zinc bacitracin.

The mix is then flattened, covered with powder, and set aside for 1 to 2 hours to permit absorption of more powder. The mix is again kneaded, covered with powder, and set aside for another 1 to 2 hours, after which it is kneaded once again. Finally, it is divided into portions, powdered, wrapped in foil, and refrigerated (but not frozen) until needed. Before use, it is unwrapped, softened by kneading, and applied to the wound. Experience has shown that the stored dressing should be used within 10 days.

Coe-Pak Dressing. Coe-Pak is another noneugenol dressing. It is supplied in two tubes from which equal lengths of material are expressed. These are mixed to a homogenous consistency and applied to the wound. Before setting, this dressing has the consistency of putty. Although it does not become rigid, it does become stiff and loses its plasticity. To increase the rigidity, some clinicians add powder from a standard eugenol dressing.

Placement

Gingivectomy/Gingivoplasty. The dressing is removed from the freezer at the beginning of the surgical procedure. In many instances, it can be manipulated by the time the surgery is finished. If not, the dressing can be softened by holding it close to the unit light or placing it in hot water. Once the dressing is soft, small pointed sections are fashioned and forced interproximally with a plastic instrument. The dressing will occupy space that might otherwise be filled by granulation tissue and, in effect, will act as a template to confine the healing tissue. Interproximal packing also aids in retention of the dressing. Next, longer strips are placed on the facial, lingual, and palatal aspects and joined to the interproximal sections. The dressing should cover the entire wound area, but should not interfere with occlusion or muscle attachments. A common error is to make the dressing too large. Silicate lubricant is used to smooth the surface of the dressing. Dry foil is often placed over the dressing to permit hardening without displacement. The patient can remove the dry foil in 6 to 8 hours if so desired.

Flaps and Grafts. Interproximal packing should be avoided after flap surgery because it is possible to push the dressing under the flap margin. It is often advantageous to cover the flap or graft with a protective material such as Stomahesive before placement of the dressing to prevent tissue displacement, to avoid wedging of dressing under the tissue, and to prevent the dressing from binding with the suture. The dressing is flattened and gently placed over the surgical site. A plastic instrument is used to work the dressing interproximally. Silicate lubricant is used to smooth the surface of the dressing.

Removal

The initial dressing is left in place for about 1 week. When it is removed, the entire area is cleansed with warm water or dilute hydrogen peroxide. Any dressing fragments found embedded in soft tissue or in interproximal areas are removed. The tooth surfaces are carefully examined, and any accumulated plaque, debris, or remaining calculus or dressing is removed. Irritants not removed during surgery or dressing changes will cause formation of excess granulation tissue. If the dressing is not replaced, the teeth are polished, and the patient is instructed in plaque control. The criterion for replacement of a dressing is whether or not the patient can accomplish plaque control without damaging the healing tissue.

Postsurgical Considerations

POSTOPERATIVE INSTRUCTIONS

Postoperative instructions to the patient can be either written or verbal. The latter may sacrifice understanding for speed and may not be remembered by the patient. Written instructions need not be elaborate and can be tailored to individuals. For example, a patient who has undergone flap surgery can be given the following instructions:

Do not be surprised or alarmed by a red tinge to your saliva for a while. If you have bleeding that gives you concern, call me.

Expect some swelling. Immediately apply ice for 20 minutes, and then discontinue for 20 minutes. Repeat this procedure 2 to 3 times during the day. This will help reduce chances of swelling.

Don't drink hot beverages during the first 4 hours or alcoholic beverages during the first 24 hours.

The prescribed medicine should control discomfort. If you have unusual pain, call me.

Avoid raw carrots, apples, nuts, and other hard foods that may dislodge the dressing. Eat well-balanced meals. If you have trouble chewing your food, drink food supplements with meals.

If the dressing becomes loose or is lost, call me.

Clean the rest of your teeth as instructed, but avoid the area with the dressing. Frequent use of a mouthwash will help control the strange taste of the dressing.

These or similar written instructions can be mimeographed for use where quantities are needed. They should be supplemented by verbal instructions on an individual basis.

POSTOPERATIVE PROBLEMS

Bleeding and loose or lost dressings are infrequent problems after surgery, but they are the commonest problems. When a patient returns because of bleeding, the dressing should usually be removed entirely. The source of bleeding should be located by gently removing any clots which conceal it. Pressure applied to the site with gauze soaked in saline solution for about 5 minutes usually stops the bleeding. If it does not, another 5-minute application is indicated. Frequently, injection of the bleeding site with an anesthetic containing 1:50,000 epinephrine will stop the bleeding. If all else fails, thrombin (5,000 N.I.H.) applied topically will usually control the bleeding. Remember that uncontrolled bleeding may be an indication of a deficiency in the bleeding or clotting mechanism, and an evaluation of these systems may be required.

If the problem is a loose dressing, the dressing should be entirely removed and a new dressing placed after cessation of any resultant bleeding.

Tense swelling, severe pain, obvious purulence, or fever and malaise indicate infection, which should be attacked promptly and vigorously. Infection rarely follows careful periodontal surgery, but if it does, penicillin or erythromycin can be used effectively.

Post operative root sensitivity may occur after dressings have been removed and usually results from ineffective plaque control. Treatment of root sensitivity is discussed in chapter 21, "Periodontal emergencies." Because of greater emphasis on plaque control and on new attachment procedures rather than on resection, root sensitivity is not as common as in years past.

Limitations of Surgery

Surgery is not curative. It is a means of providing access and of restoring missing parts of the periodontium. When performed for motivated, cooperative patients by skilled, knowledgeable practitioners, it is an important part of periodontal treatment.

14 Management of soft tissue: subgingival curettage and ENAP

Pocket elimination
- **Shrinkage**
- **Resection**
- **New attachment**
- **Movement of the soft tissue wall apically**

Subgingival curettage
- **Indications**
- **Objective**
- **Technique**

Excisional new attachment procedure (ENAP)
- **Indications**
- **Objective**
- **Technique**

Pocket Elimination

The periodontal pocket is a space bounded by the tooth on one side and by ulcerated epithelium lining the soft tissue wall on the other. Its base is the epithelial attachment. The pocket can be eliminated by removing the tooth surface (extraction or root removal) or by doing something to the pocket wall.

There are four general approaches to management of the soft tissue wall and elimination of the space:

1. Shrinkage of the soft tissue.
2. Resection of the soft tissue wall.
3. New attachment of soft tissue to teeth.
4. Movement of the soft tissue wall apically.

SHRINKAGE

Removal of plaque, calculus, and exposed cementum by scaling and root planing, followed by the establishment of plaque control, will reduce inflammation and effect shrinkage of the tissue. Occasionally, the amount of shrinkage is astonishing. Many areas initially considered certain to require further treatment by surgery are later found to require no additional treatment.

RESECTION

Resection of the soft tissue wall is accomplished by gingivectomy, which will be discussed in

chapter 15, "Management of soft tissue: gingivoplasty and gingivectomy."

NEW ATTACHMENT

New attachment implies formation of new cementum, connective tissue fibers, epithelial attachment, and in some cases bone. Many surgical procedures result in new attachment of tissue to tooth during healing, but this attachment is often at, or apical to, the preexisting level. To achieve new attachment at a more coronal level, certain procedures must be accomplished during surgery:

1. All pocket and attachment epithelium must be removed.
2. Cementum in the environment of the pocket must be planed.
3. Apposition of tissue to tooth must be as close as possible, facially, lingually, and proximally. The smaller the clot, the more quickly granulation tissue cells can approach the tooth and produce the needed support elements.
4. Primary wound closure must be obtained.

Two surgical procedures specifically aimed at establishing a new connective tissue attachment to the tooth at a more coronal level than existed preoperatively are subgingival curettage and the excisional new attachment procedure (ENAP).

MOVEMENT OF THE SOFT TISSUE WALL APICALLY

Apical movement of the soft tissue wall of the pocket will predictably eliminate the defect. This technique is described in chapter 16, "Management of soft tissue: flaps and free grafts."

Subgingival Curettage

INDICATIONS

The classic indications for subgingival curettage are the presence of shallow pockets and an adequate width of gingival tissue that is swollen, inflamed, and nonfibrotic.

OBJECTIVE

The objective of this procedure is pocket elimination by shrinkage and new attachment

TECHNIQUE

The procedure is performed under local anesthesia. The objective is to scale and root plane the tooth surfaces, being careful to remove loosened calculus completely from the pocket, and then to peel out the epithelial lining and granulation tissue with a sharp curet. When bleeding stops and clots are established, a surgical dressing is used to cover the area.

Subgingival curettage with a curet is possibly talked about and written about more often than it is actually achieved. First, it is a somewhat demanding technical procedure, requiring very sharp curets and great finesse. Second, it is a rather crude surgical procedure that macerates tissue, requiring further clean-up by the cells during healing. Third, root visualization is poor unless the pockets have been dilated by leaving some kind of "packing" in them for a few days preoperatively.

It is said that a new attachment is formed during healing. Although this may occur, the reduction in pocket depth can usually be explained as the result of the removal of tissue during curettage and shrinkage afterwards. If shrinkage by resolution of inflammation is desired, this can be attained by removal and prevention of causative agents. If diminution in bulk is desired, this can be achieved by surgical procedures less traumatic and more predictable than the comparatively blind scraping with a curet.

Nevertheless, deliberate subgingival curettage can sometimes be a useful technique in periodontal treatment. Isolated pockets (e.g., a deep pocket on the palatal aspect of a maxillary cuspid), whether the tissue is fibrosed or not, may warrant an attempt at subgingival curettage. If enough care is taken to scale and root plane thoroughly, to rinse the defect as clean as possible, to scrape out all of the epithelial lining and epithelial attachment, and to maintain pressure (2 to 3 minutes) against the gingiva after all other procedures have been accomplished (to ensure as small a clot as possible), then conditions will have been established to permit new attachment during healing.

Excisional New Attachment Procedure (ENAP)

In essence, ENAP is subgingival curettage performed with a knife. The inner aspect (epithelium and some connective tissue) of the periodontal pocket is excised; and the remaining gingival tissue is closely approximated against and between the teeth, permitting formation of a new attachment during healing.

INDICATIONS

The ENAP is indicated:

1. When there is shallow to moderate pocket depth coronal to the mucogingival junction. It is particularly indicated in the anterior region, where esthetics is a consideration.

2. When there is an adequate band of keratinized gingiva. ENAP does not increase the width of keratinized gingiva. Consequently, if there is a narrow band of keratinized gingiva to start with, there will likewise be a narrow band of keratinized gingiva after excisional new attachment.

3. When there is interproximal pocket depth but minimal facial or lingual pocketing. Resection and certain flap techniques would destroy the sound connective tissue attachment to the tooth on the facial and lingual surfaces in the process of eliminating the interproximal pockets.

OBJECTIVE

The objective of ENAP is pocket elimination by establishing a new connective tissue attachment to the tooth at a more coronal level. There is little question that some shrinkage occurs in this surgical procedure, but clinical studies also indicate a more coronal attachment of soft tissue.

TECHNIQUE

Figures 14-1, 14-2, and 14-3 illustrate the ENAP technique.

Once the patient has established plaque control and initial preparation is complete:

1. Anesthetize the area.

2. Measure pocket depth with a probe, and penetrate the gingival tissue at this distance with the probe (fig. 14-1a).

3. Make a reverse bevel (internally beveled) incision with a surgical blade from the margin of

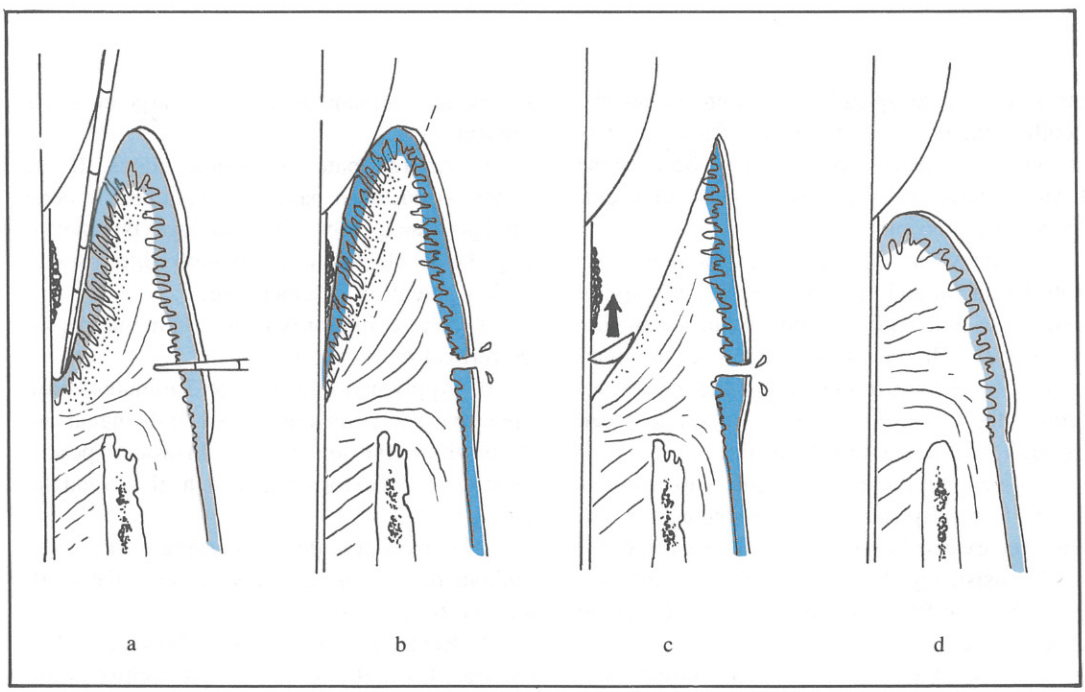

Figure 14-1

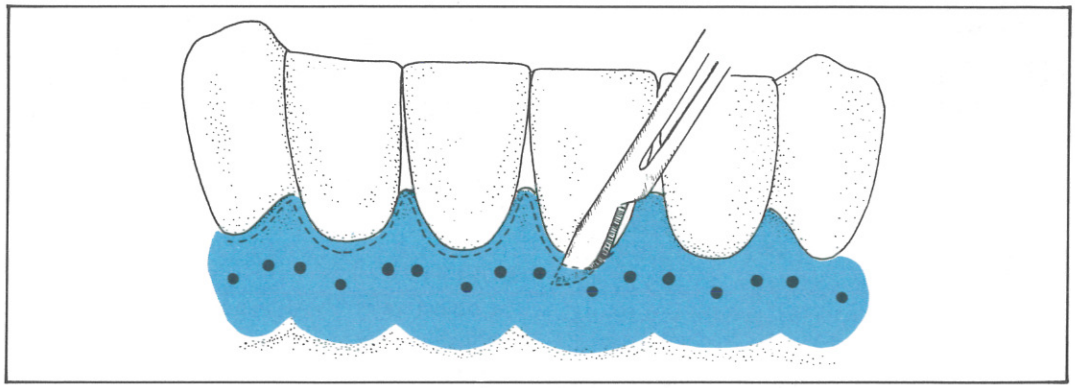

Figure 14-2

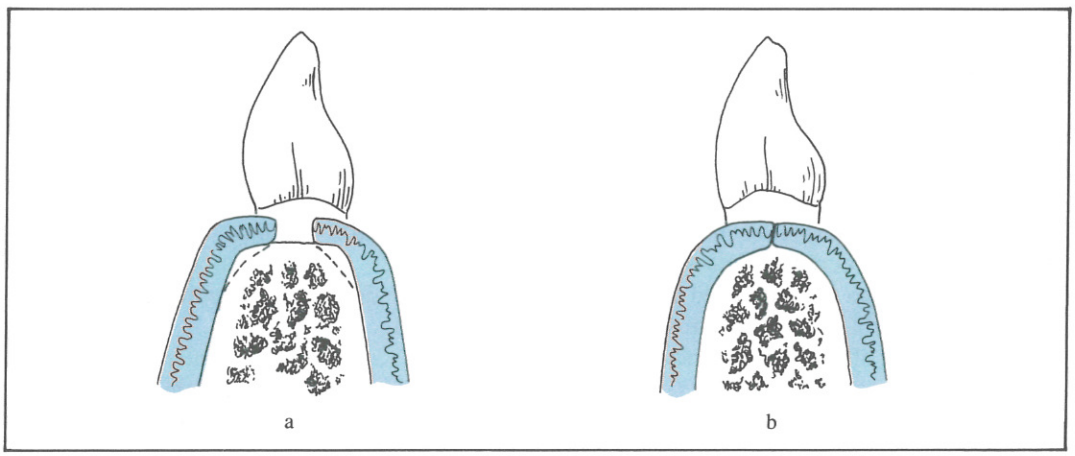

Figure 14-3

the free gingiva apically to a point below the depth of the pocket (fig. 14-1b). The incision is sometimes termed "*reverse bevel,*" because the tissue is beveled exactly the reverse of a gingivectomy incision.

4. Carry the incision interproximally on both the facial and the lingual sides, attempting to retain as much interproximal tissue as possible (fig. 14-2). The intention is to cut out the inner portion of the soft tissue wall of the pocket, all around the tooth. No attempt is made to break through the mucogingival junction.

5. Remove the excised tissue with a curet.

6. Carefully root plane all cementum that has been exposed to the oral cavity to a smooth, hard consistency (fig. 14-1c). Preserve all connective tissue fibers that remain attached to the root surface.

7. Rinse the area with normal saline solution, and examine the root surface to ensure that no calculus remains and that no large clots are present.

8. Approximate the wound edges. If the edges do not meet passively, contour the bone until good adaptation of wound edges is achieved (fig. 14-3). Healing of the wound edges by secondary intention is imperative.

9. Suture interproximally with interrupted or vertical mattress sutures.

10. Apply pressure for 2 to 3 minutes to the operative site from both facial and lingual aspects with saline-soaked gauze in order to permit only a thin clot to form between the tissue and the tooth.

11. Place a periodontal dressing over the site without forcing the dressing between the tooth and the tissue.

12. Remove the sutures in 7 days and polish the area. Carefully review plaque control of the area with the patient. Advise him/her to brush

and floss the area carefully but meticulously. Success is dependent on the patient's ability to control plaque during the critical first 3 to 4 weeks of healing.

13. Do not probe for 3 months in order to permit complete attachment of connective tissue fibers.

The ENAP is a comparatively simple surgical procedure whose objective is to eliminate the pocket by establishing a new connective tissue attachment at a more coronal level (fig. 14-1d). Clinical experience has demonstrated the technique to be a predictable and effective method of eliminating pockets.

15 Management of soft tissue: gingivoplasty and gingivectomy

Gingivoplasty
Indications
Technique
Gingivectomy
Indications
Technique

The chief purpose of gingivoplasty is the restoration of physiologic gingival contours that will help prevent recurrence of periodontal disease. The restoration of an esthetic appearance is also an important consideration. On the other hand, the basic purpose of gingivectomy is the elimination of a pocket. Both procedures should result in increased access for plaque control by the patient.

Gingivoplasty

INDICATIONS

Gingivoplasty entails the beveling of the gingival margin or interdental papillae and the creation of interdental spillways by blending the architecture of the interdental papillae with the interdental grooves. Gingivoplasty is usually indicated for tissues that are firm, fibrotic, and easily incised and contoured. This type of tissue most frequently results from chronic irritation.

TECHNIQUE

Gingivoplasty can be performed with a periodontal knife or coarse diamond stones:

1. When a periodontal knife, such as the Kirkland No. 15 or No. 16, is used, the tissue is excised to establish the basic contours. The knife is then used to scrape the tissues to achieve the final gingival architecture (fig. 15-1).

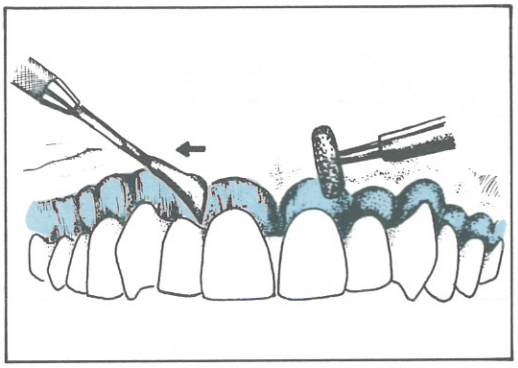

Figure 15-1

2. Coarse diamond stones may also be used (fig. 15-1). The stones may be of various shapes, depending on the need and preference of the clinician (fig. 15-2). A steady stream of sterile saline solution or sterile water must be used to prevent burning of the tissue and clogging of the stone. If osseous contouring is indicated, gingivoplasty and osteoplasty can be accomplished simultaneously with diamond stones. When stones are used, the soft tissue will often show minute shreds or tags, which can be removed with fine scissors or nippers.

After the gingiva has been contoured by either of the gingivoplasty techniques described, a periodontal dressing is placed over the area. The dressing is changed weekly until sufficient healing has occurred to permit plaque control by the patient. At each dressing change, the operator should gently remove any accumulated plaque with floss, tape, or yarn proximally and with a curet or polishing cup facially and lingually, avoiding damage to the healing tissue. At the time of dressing removal, all teeth are polished and the patient is reinstructed in plaque control procedures.

Gingivectomy

INDICATIONS

Gingivectomy is the excision of the gingival walls of periodontal pockets. The procedure is indicated for the elimination of periodontal pockets when excision of the pocket wall will not result in an inadequate zone of attached gingiva.

Figure 15-2

TECHNIQUE

1. As the first step in gingivectomy, obtain satisfactory anesthesia, by either the block or the infiltration technique. A great aid in obtaining hemostasis is to inject lidocaine hydrochloride 2% with 1:50,000 epinephrine in the immediate area of the operation.

2. Measure the pocket depths with a calibrated probe and mark the level by puncturing the outer wall of gingival tissue, to establish bleeding points. When the entire area has been adequately measured and marked, the bleeding points will outline the required incision.

3. Make the initial incision apical to the bleeding points with a broad-bladed knife, such as the Kirkland No. 15 or No. 16 (fig. 15-3). The incision should be beveled to about a 45° angle to the tooth. Where the gingiva is thick, emphasize the bevel by lengthening, to eliminate a plateau or shoulder. Access may be limited or difficult so that a proper bevel cannot be obtained with the initial incision. In this case the bevel can be corrected later, either with a broad-bladed knife used as a scraper, or with coarse, abrasive rotary diamond stones.

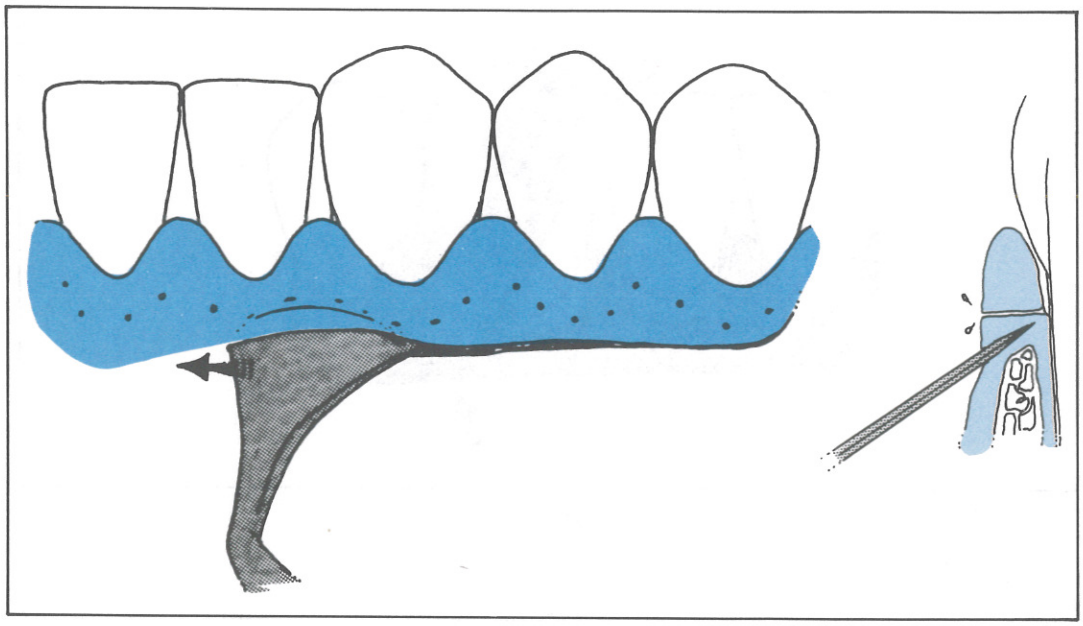

Figure 15-3

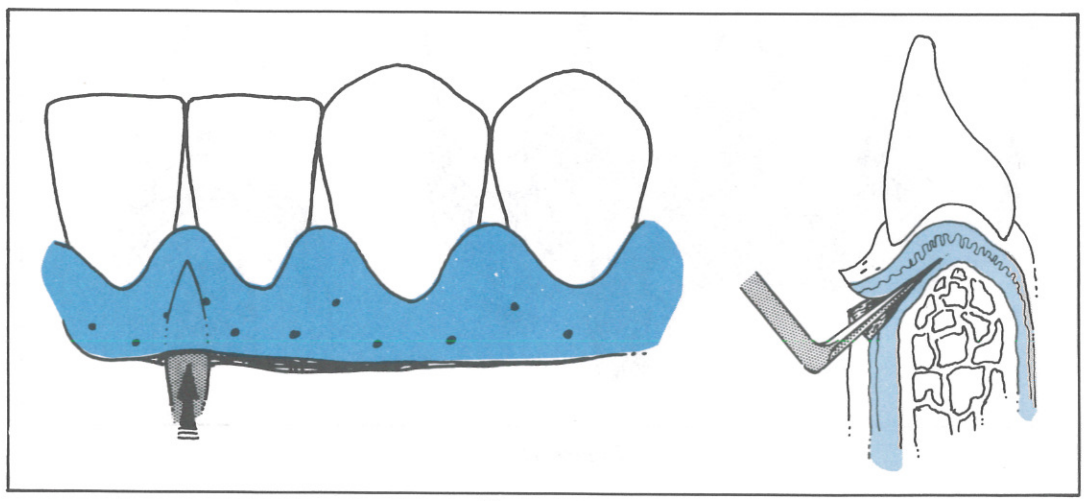

Figure 15-4

4. Use narrow-bladed knives, such as the Orban No. 1 and No. 2, to incise the tissue interproximally (fig. 15-4).

5. Remove the incised gingival tissue with curets (fig. 15-5).

6. Remove accretions from the root surfaces. Removal of the soft tissue walls of periodontal pockets renders these surfaces more accessible and more visible to the operator now than at any other time. Success or failure of the entire procedure depends on how well the operator performs scaling and root planing.

7. Complete further contouring as needed, by using coarse diamond stones, or a broad-bladed knife to scrape the tissue (fig. 15-6).

8. Remove tissue tags with scissors.

9. Flush the surgical site with sterile saline solution to remove foreign particles.

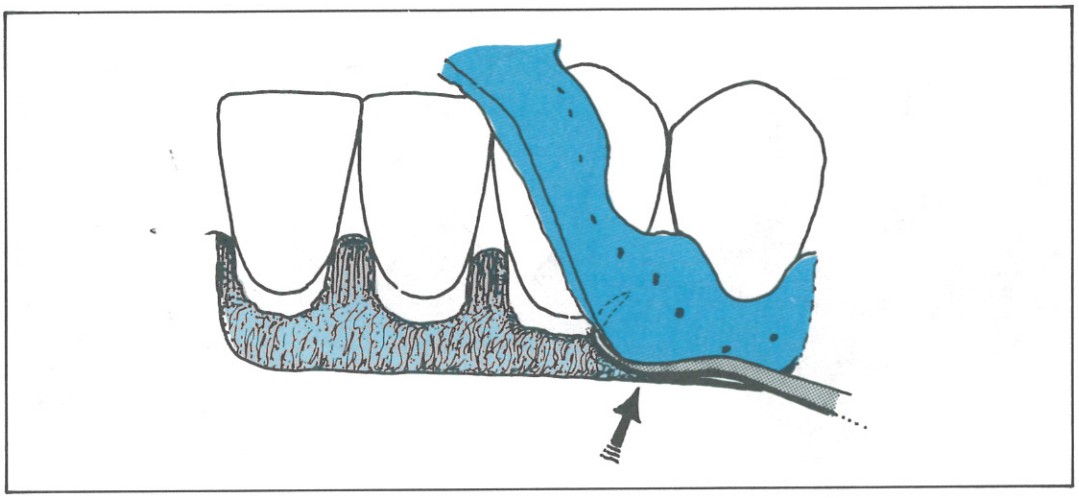

Figure 15-5

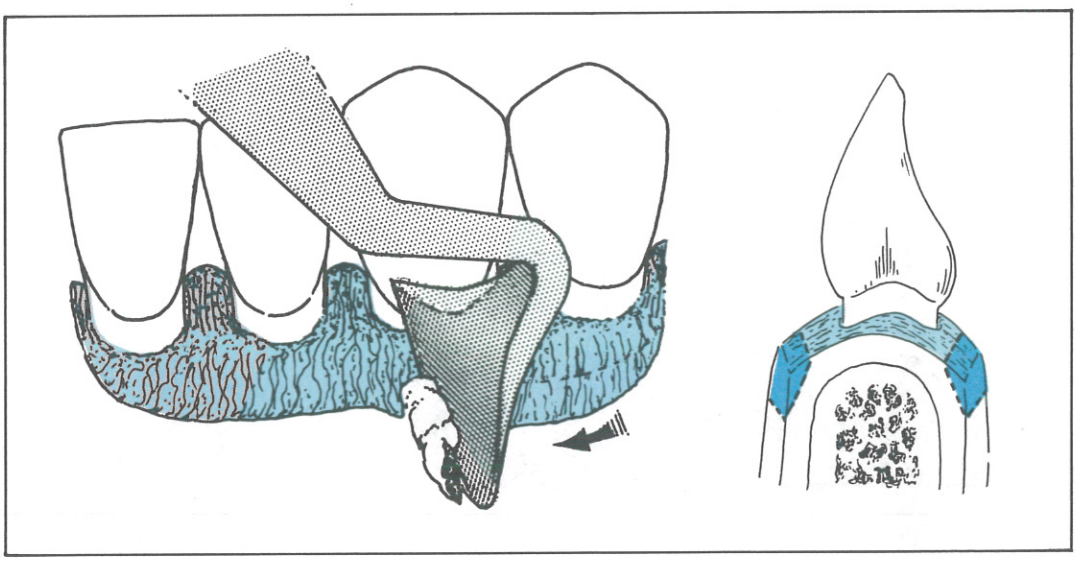

Figure 15-6

10. Apply constant pressure against the wound for 2 to 3 minutes, with gauze squares saturated with saline solution, to stop bleeding.

11. Apply periodontal dressing by initially placing small pointed sections interproximally with a plastic instrument. Next, place longer strips on the facial, lingual, and palatal aspects and join them to the interproximal sections. Cover the entire wound area with the dressing, taking care not to let it interfere with occlusion or muscle attachments. A common error is to make the dressing too large.

12. Change the dressing weekly until the tissues have healed sufficiently for the patient to accomplish plaque control.

13. After the dressing is removed, polish the teeth and instruct the patient in plaque control.

16 Management of soft tissue: flaps and free grafts

Basic concepts and considerations
- Objectives of flaps and free grafts
- Classification of flaps
- Partial-thickness flap
- Full-thickness flap
- Partial-thickness or full-thickness flaps
- Vertical relaxing incisions

Flaps and free grafts
- The replaced flap
  - Indications
  - Technique
- The apically positioned flap
  - Indications
  - Technique
- The laterally positioned flap
  - Indications
  - Technique
- The double papillae flap
  - Indications
  - Technique
- Free gingival graft (free soft tissue autograft)
  - Indications
  - Technique

Management of other soft tissue problems
- Aberrant frena (frenectomy)
- Shallow vestibule (vestibuloplasty)
- Deep pocket retromolar area (distal wedge procedure)

A summary of management of common soft tissue problems

Basic Concepts and Considerations

OBJECTIVES OF FLAPS AND FREE GRAFTS

Generally, flap and free grafting techniques are designed to accomplish one or more of the following:

1. Eliminate pockets that extend to or below the mucogingival junction.
2. Create an adequate zone of attached gingiva.
3. Permit access to underlying bone for osseous surgery and grafting.
4. Eliminate gingival clefts.
5. Relieve the pull of frena and muscle attachments on the free gingival margin.
6. Establish new connective tissue attachment at a more coronal level.

CLASSIFICATION OF FLAPS

A flap is defined as that portion of the gingiva, alveolar mucosa, or periosteum which is elevated or dissected from the alveolar process and which retains its blood supply. Flaps may be classified on the basis of (1) tissue components and (2) disposition of these components at the completion of surgery. The following classification of the more commonly employed techniques is used at the Naval Graduate Dental School:

1. *Partial-thickness flap:* flap that is composed of mucosa or mucosa and submucosa. A partial-thickness flap may be:
   a. Replaced.
   b. Apically positioned.
   c. Laterally positioned.

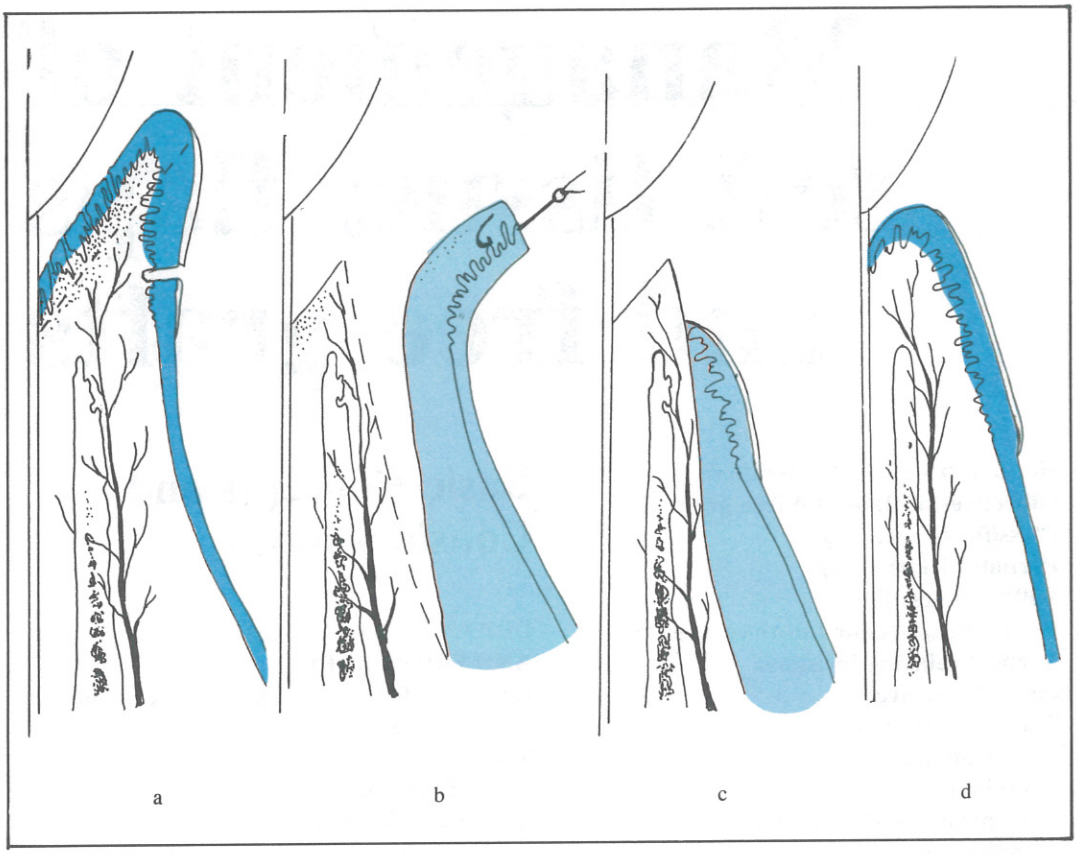

Figure 16-1

2. *Full-thickness flap:* flap that is composed of mucosa, submucosa, and periosteum. A full-thickness flap may be:

a. Replaced.
b. Apically positioned.
c. Laterally positioned.

Flaps can be described as apically positioned partial-thickness flaps, replaced full-thickness flaps, laterally positioned full-thickness flaps, etc.

PARTIAL-THICKNESS FLAP

The partial-thickness flap is prepared by sharp dissection close to bone with the intent of leaving some connective tissue attached to the bone. The technique is as follows:

1. Make a scalloped, reverse bevel incision from the surface of the tissue to the base of the periodontal pocket (fig. 16-1a). Bard-Parker blades Nos. 11, 12-B, or 15 can be used on both the facial and the lingual aspect. Use of one of the aforementioned blades with an ASR handle may provide better angulation on the lingual aspect.

2. Remove the excised tissue with a curet.

3. Make a second incision close to bone leaving soft tissue attached to bone (fig. 16-1b). The flap is easily dissected if light tension is placed on the coronal portion of the flap. This can be accomplished by placing a suture or Hirschfeld hook through the flap to aid in retraction (fig. 16-1b).

4. Thoroughly scale and plane the roots. Care is taken not to remove connective tissue fibers attached to the roots.

5. Depending on the surgical site and the surgical objective, replace the flap now in its original position, apically positioned or laterally positioned. Figure 16-1c shows the flap apically positioned.

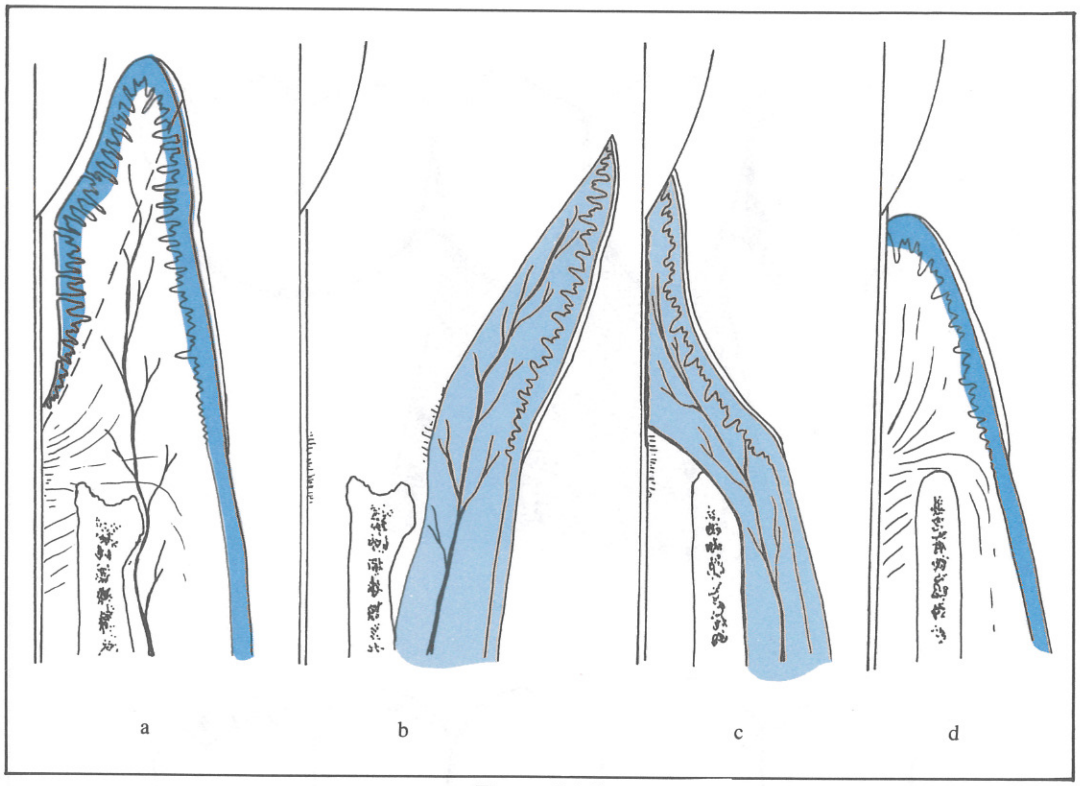

Figure 16-2

6. Healing is schematically represented in figure 16-1d.

FULL-THICKNESS FLAP

The full-thickness flap is prepared by bluntly dissecting soft tissue from the bone with a periosteal elevator. It is performed as follows:

1. Make a scalloped reverse bevel incision to the base of the periodontal pocket, preserving as much keratinized gingiva as possible. Bard-Parker blades Nos. 11, 12-B, or 15 are commonly used to make this incision (fig. 16-2a).
2. Bluntly dissect the tissue with a periosteal elevator to the desired depth for access or for flap mobility (fig. 16-2b).
3. Remove the excised tissue with a curet.
4. Scale and plane the roots.
5. Depending on the surgical site and the surgical objective, the flap can be replaced in its original position, apically positioned, or laterally positioned. In figure 16-2c, the flap is replaced in its original position.
6. Healing is represented in figure 16-2d.

PARTIAL-THICKNESS OR FULL-THICKNESS FLAPS

There is considerable controversy regarding the routine use of either a partial-thickness or a full-thickness flap. Some clinicians believe that bone loss is less likely to be permanent if partial-thickness flaps are used. Others have shown that full-thickness flaps actually result in less bone loss. Advocates of full-thickness flaps are quick to point out that there is greater likelihood of necrosis of wound edges of the partial-thickness flap due to reduced blood supply. Also, surgical perforation is more likely in the partial-thickness flap. This could result in loss of tissue, delayed healing, and would certainly jeopardize new attachment procedures. In spite of some of the apparent disadvantages of a partial-thickness flap, it is believed by many that this flap is indicated in any of the following situations:

1. Tissue is removed laterally or horizontally from an adjacent tooth.
2. A bony dehiscence or fenestration is suspected.

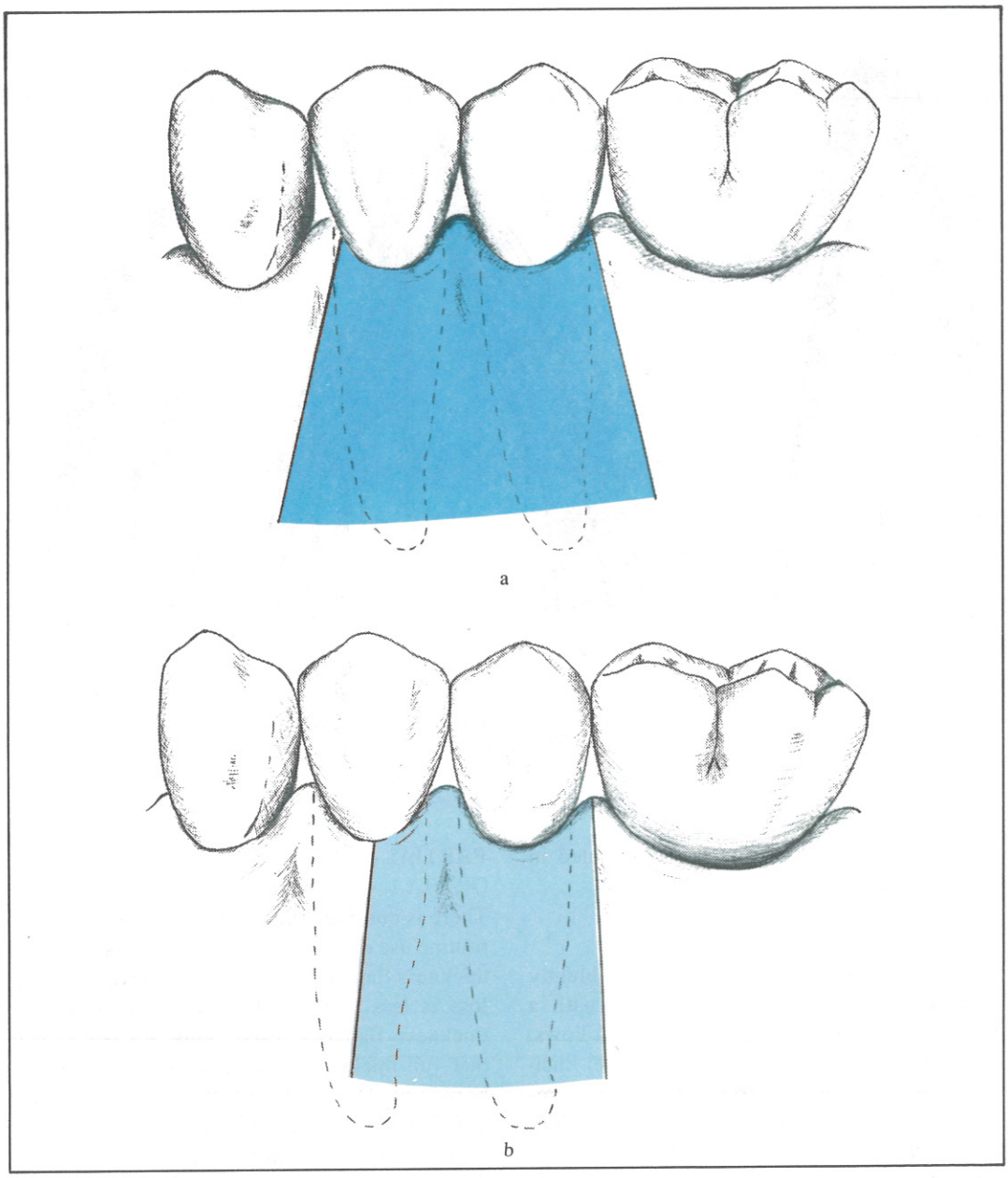

Figure 16-3

3. Radicular bone is known to be thin, and a sufficient thickness of soft tissue permits dissection without flap perforation or severance of the blood supply to the coronal edge of the flap.

4. There is bulbous or hyperplastic gingiva, as is often the case on the palate.

VERTICAL RELAXING INCISIONS

Vertical relaxing incisions may be employed on the facial surface when access and visibility are limited (fig. 16-3a). They should be made at line angles to preserve the interdental papillae for suturing and to prevent necrosis of the wound edge. Under no circumstances should vertical incisions be made over the midfacial (radicular) surface of roots (fig. 16-3b). The vertical incision can also aid the clinician in preparing a partial-thickness flap. Once the vertical incision is made, a Bard-Parker No. 15 blade is inserted horizontally into the loose tissue at the mucosal

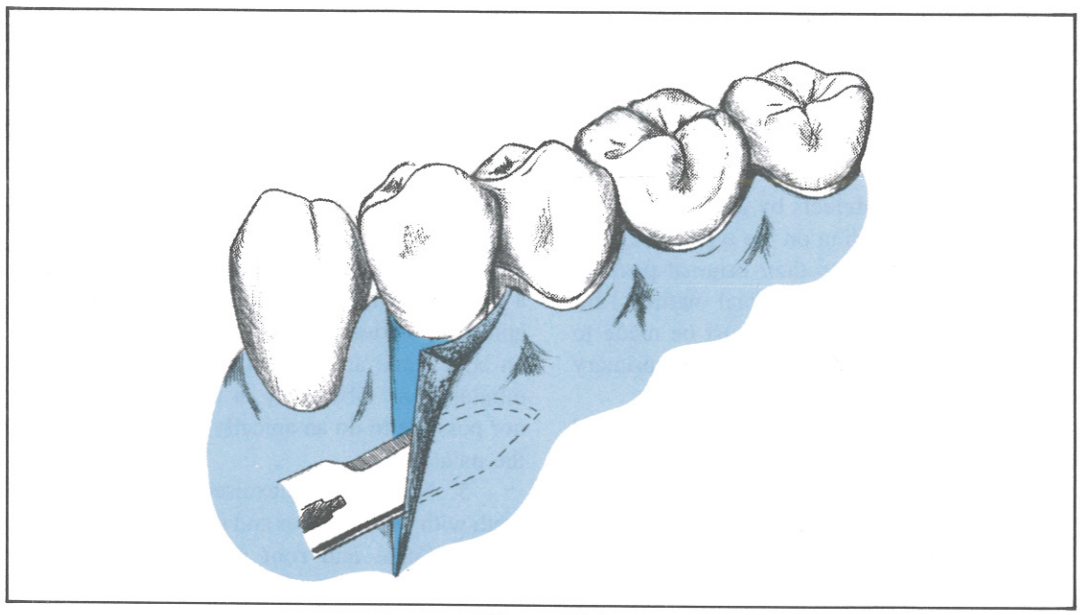

Figure 16-4

end of the incision and the tissue is dissected in a coronal direction (fig. 16-4). There is much less likelihood of perforation or detachment of the gingiva when partial-thickness dissection is performed in this manner. However, vertical relaxing incisions should not be performed indiscriminately. There are certain disadvantages associated with them, such as: (1) a greater opportunity for undesirable flap displacement when relaxing incisions are utilized and (2) an increase in the likelihood of postoperative swelling, bleeding, and patient discomfort. Also, vertical incisions are not recommended on the palate and the lingual surfaces, because of surgical and postoperative complications of bleeding and infection, respectively.

The flap procedures most frequently utilized in periodontal surgery are the replaced flap; the apically positioned flap; the laterally positioned flap; and the double papillae flap, a modification of the laterally positioned flap.

Flaps and Free Grafts

THE REPLACED FLAP

Indications

A replaced flap is one which is placed back near or at its original position. It is used:

1. To gain access to underlying bone and root surfaces.
2. To eliminate pockets by establishment of a new connective tissue attachment at a more coronal level.

The replaced flap is contraindicated if there is an inadequate zone of keratinized gingiva. In this instance, a technique such as the apically positioned flap is indicated, not only to increase the width of gingiva but also to dissipate the pull of the connective tissue and muscle fibers.

Technique

(Refer to figure 16-2.)

1. Mark the depth of the pockets in order to create reference points.
2. Make a reverse bevel incision around the necks of the teeth with a No. 11, 12-B, or 15 blade. The No. 11 blade in an ASR handle works well on the lingual or palatal surfaces. Direct the incision toward the base of the pocket.
3. Make a scalloped incision around the necks of the teeth and interproximally, in order to preserve interproximal tissue for primary wound closure.
4. Elevate a full-thickness flap by blunt dissection or a partial-thickness flap by sharp dissection. Occasionally, one or more relaxing incisions may be needed to provide improved access.

5. Root plane the involved teeth to a smooth, hard consistency. Remember, soft tissue fibers attached to the root surface should be left attached. Only the root surfaces which had been exposed to the environment of the pocket need be treated.

6. Treat bony defects by grafting or osseous resection, depending on the type of defect.

7. Replace flaps to their original position and suture (interrupted or vertical mattress) interproximally. Every attempt must be made to approximate wound edges and obtain primary wound closure.

8. Apply firm but gentle pressure for 2 to 3 minutes on the facial and lingual flaps with gauze moistened in sterile saline solution.

9. Some clinicians apply Stomahesive to prevent wedging of dressing beneath the flap and to prevent the dressing from binding to the sutures.

10. Remove sutures on the seventh day and carefully polish the area. Instruct the patient in plaque control of this area.

THE APICALLY POSITIONED FLAP

Indications

An apically positioned flap is one that is placed apical to its original position at the end of the procedure. It is used:

1. To eliminate pockets by repositioning the gingival tissue apically.

2. To increase the zone of attached gingiva. This is accomplished by moving existing mature gingiva apically on the alveolar process. Collagen fibers from the periodontal ligament and from the granulation tissue will form coronal to the margin of apically positioned gingiva. When mature, this tissue will function as attached gingiva.

Technique

(Refer to figure 16-1.)

1. Make a reverse bevel incision through the gingiva to the depth of the pocket.

2. Since interproximal apposition of wound edges is not feasible, it is unnecessary to retain as much interproximal tissue as in the excisional new attachment procedure (ENAP) or the replaced flap procedure. However, a scalloped incision is preferable in order to retain some keratinized gingiva on the wound edge.

3. Reflect a full- or partial-thickness flap. It is not uncommon to begin by reflecting a full-thickness flap and then change to partial thickness to achieve flap mobility.

4. Perform whatever surgery is indicated, on the opposite side of the teeth. The procedure is usually combined with either a replaced flap or a gingivectomy on the opposite side, although apically positioned flaps are occasionally done on both facial and lingual surfaces of the mandibular teeth. Obviously, it is neither necessary nor possible to do an apically positioned flap on the palate.

5. Next, free the excised tissue around the teeth with Orban knives and remove with a curet.

6. Scale and root plane the teeth to a smooth, hard consistency.

7. Perform osseous resection, if indicated.

8. Suture the margin of the keratinized gingiva at or near the crest of the alveolar process. The sutures may be tied through the tissue on the other side (interrupted) or tied around the teeth, suspending the flap at the desired position. The most common error is to suture too tightly, pulling the flap coronally and defeating the purpose of the procedure.

9. As in all flap and graft procedures, apply pressure with a gauze moistened with saline solution to ensure minimum clot formation between flap and underlying tissue.

10. Apply the dressing. Care should be taken to avoid forcing dressing under the flap. The flap margin and adjacent tissue can be covered with Stomahesive to prevent dressing from entering.

THE LATERALLY POSITIONED FLAP

Indications

The laterally positioned flap is a technique used to move gingiva from an adjacent tooth or edentulous area to a prepared recipient site, where it is grafted to the underlying tissue. It is indicated in the following situations:

1. The defect interferes with the ability to accomplish effective plaque control (fig. 16-5).

2. Esthetics is an important factor.

3. There is sufficient width of keratinized gingiva at the donor site and there are no pockets on the donor tooth or teeth.

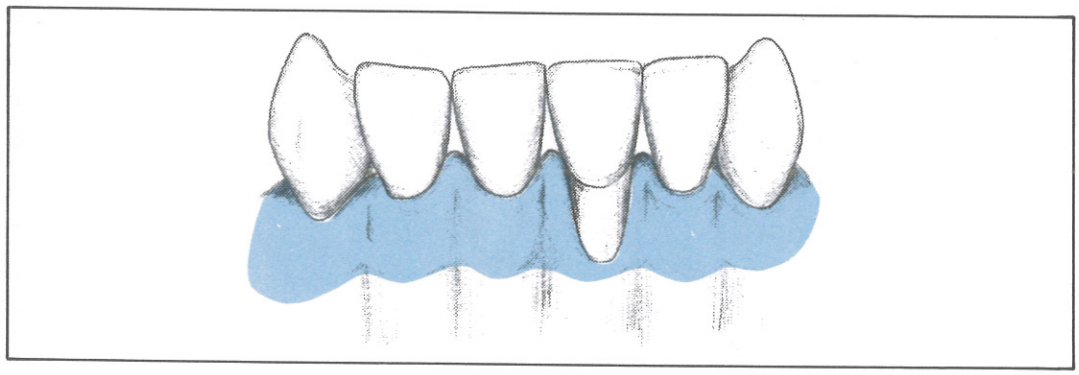

Figure 16-5

Technique

1. Make a U-shaped incision with a No. 15 blade and create a beveled wound edge around the recipient site (fig. 16-6). The wound edge to be sutured must be over bone.

2. Remove the incised tissue with a curet and root plane the cementum to a smooth, hard consistency.

3. Make a vertical incision at a distance of at least one and a half times the measurement of the recipient site (fig. 16-7).

4. Perform a partial-thickness dissection as shown in figure 16-8 to free the flap of tissue. Connective tissue must be left over the radicular surface of the donor tooth. If this is not done, it is not uncommon to observe root exposure at the donor site where none existed before. If the donor site is an edentulous area, either a partial-thickness or full-thickness flap can be elevated.

5. Position the flap at the recipient site to completely cover the defect. If there is tension on the flap as the lip or cheek is extended, make a cutback incision in the direction in which the flap is to be moved (fig. 16-9). Care must be taken not to completely sever the blood supply of the pedicle when performing this cutback procedure.

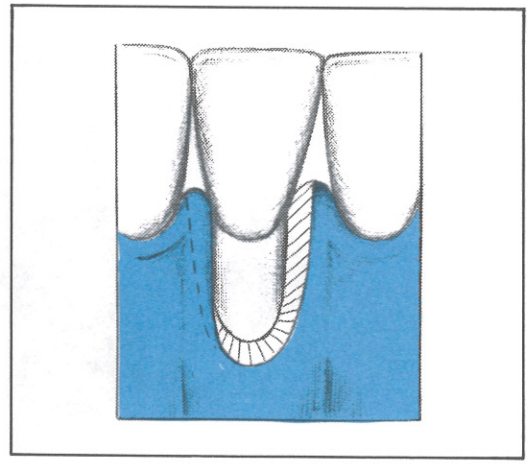

Figure 16-6

6. Suture the flap at the recipient site as shown in figure 16-10. Place interrupted sutures

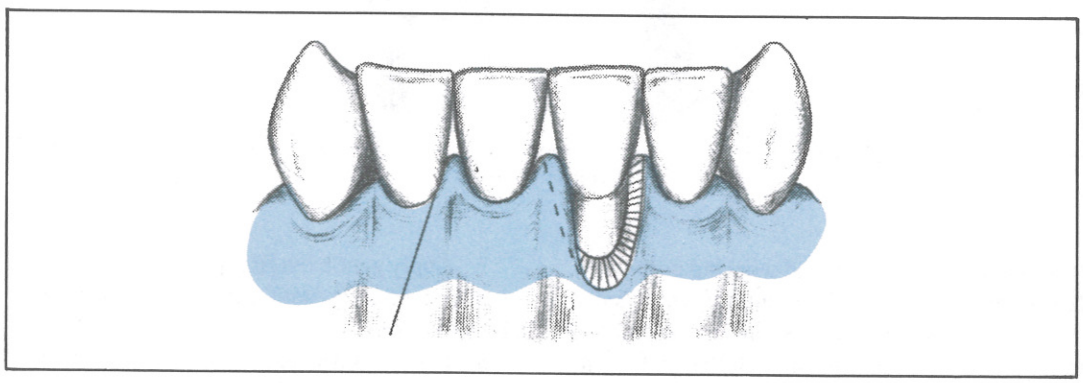

Figure 16-7

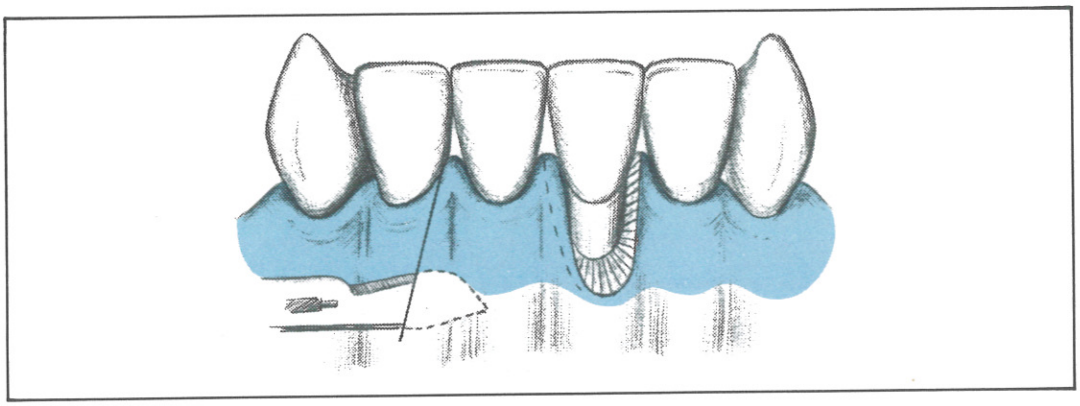

Figure 16-8

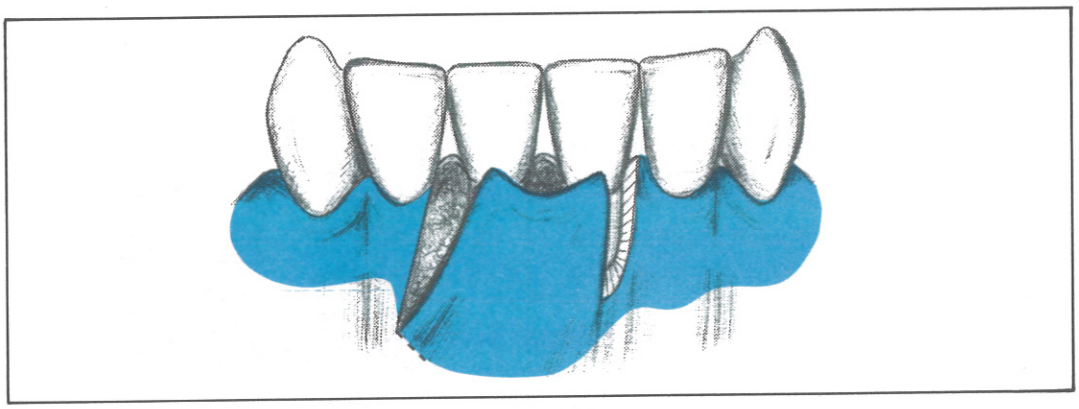

Figure 16-9

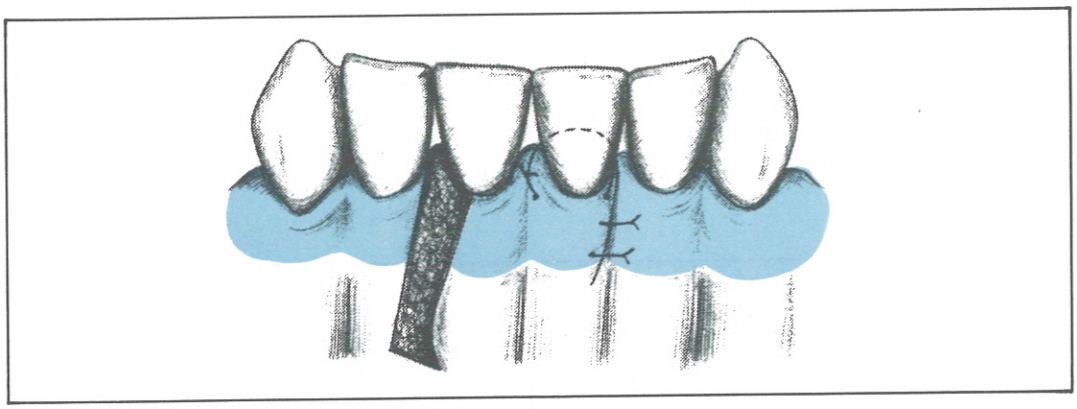

Figure 16-10

(6-0) beginning apically and working coronally. No more than two or three sutures are usually necessary. A sling suture is carried around the tooth and tied facially to prevent the graft from slipping apically.

7. Apply gentle but firm pressure to the graft for 2 to 3 minutes with gauze moistened with saline solution.

8. Cover with Stomahesive and apply periodontal dressing. The dressing must not dis-

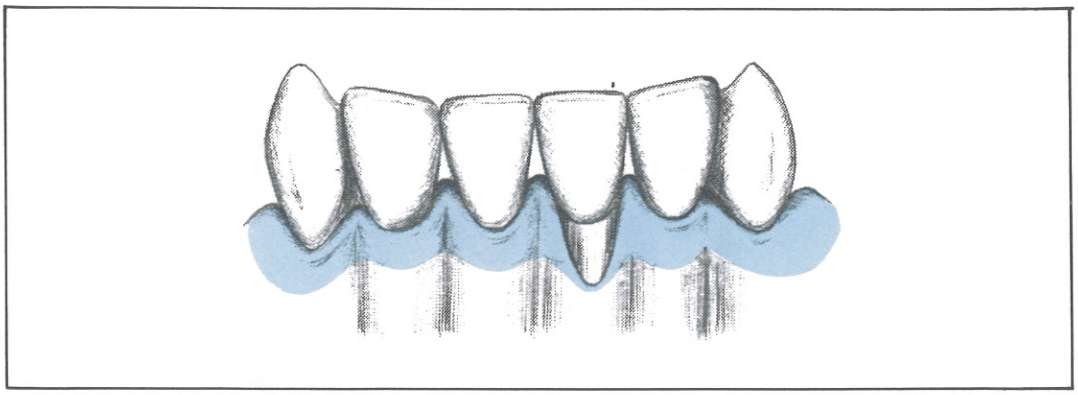

Figure 16-11

place the graft or impinge on the base of the flap. The latter action would impede the blood supply to the coronal part of the flap and result in necrosis and graft failure.

9. Remove the sutures in 7 days; polish the area; and instruct the patient in plaque control. The area should not be probed for 3 months.

When properly performed, the laterally positioned flap is a predictable surgical procedure for the repair of gingival clefts when there is sufficient width of keratinized gingiva at the donor site.

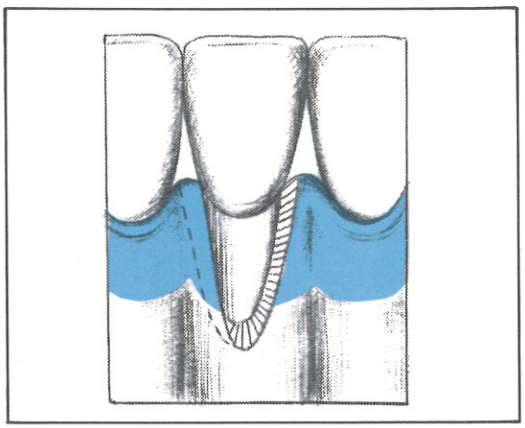

Figure 16-12

THE DOUBLE PAPILLAE FLAP

Indications

The double papillae flap is a modification of the laterally positioned flap. It can be used to repair gingival clefts when there is an adequate amount of healthy interproximal tissue adjacent to the recipient site and minimal keratinized gingiva over the radicular surfaces (fig. 16-11).

Technique

1. Make a V-shaped incision around the cleft with a No. 15 blade. Both edges of the incision should be beveled as shown in figure 16-12 to permit overlap of the connective tissue surfaces. This will enhance union of the flap margins over the avascular root surface.

2. Remove the incised tissue with a curet.

3. Scale and plane the root surface to a smooth, hard consistency.

4. Make an oblique incision along the line angles of the adjacent teeth with a No. 15 blade. Extend the incisions into the alveolar mucosa (fig. 16-13).

5. Free the papillae by blunt dissection with a periosteal elevator, or create partial-thickness flaps with a No. 15 blade.

6. Position the flaps (papillae) at the recipient site. If there is tension or movement of the flaps when the lips, or cheek, are extended, make a cutback incision to release this tension.

7. Suture the two flaps in the midline of the tooth with 6-0 suture. Begin interrupted sutures at the base of the flaps. The most coronal suture is placed circumferentially around the tooth and tied facially to hold the margin of the combined flaps on the enamel (fig. 6-14).

8. Apply gentle but firm pressure for 2 to 3 minutes with gauze moistened with saline solution.

9. Dress the area with Stomahesive and cover with periodontal dressing. Be careful not to displace the flaps with the dressing or to per-

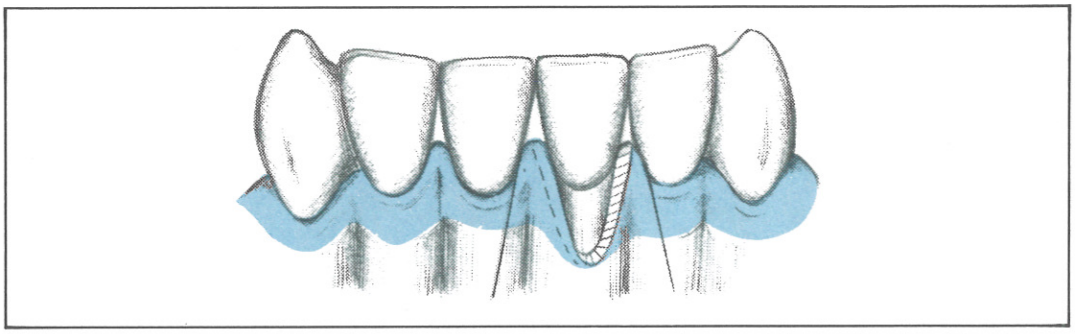

Figure 16-13

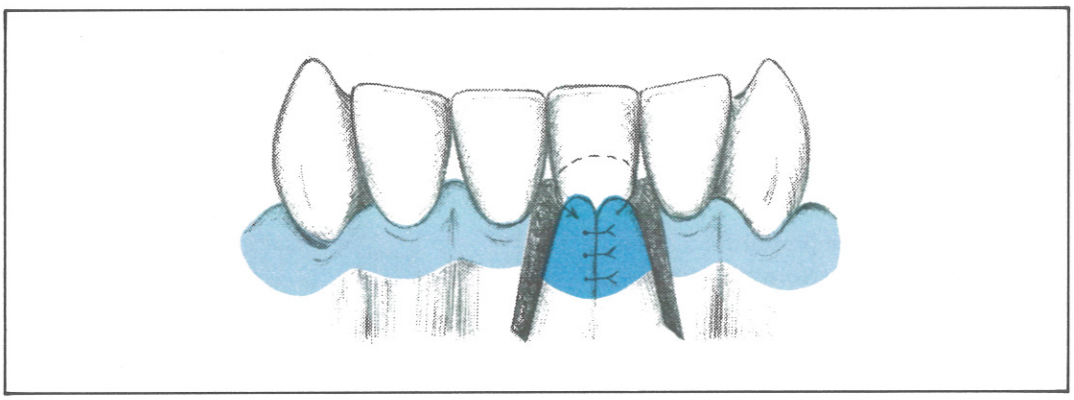

Figure 16-14

mit the dressing to impinge on the blood supply of the flaps.

10. Remove the sutures in 7 days, polish the teeth, and instruct the patient in plaque control.

FREE GINGIVAL GRAFT (FREE SOFT TISSUE AUTOGRAFT)

Indications

The development of the surgical technique for utilization of the soft tissue autograft is one of the major advances in periodontal surgery in the past decade. It is an extremely versatile and highly predictable technique. It is used:

1. To increase the zone of attached gingiva.
2. To eliminate aberrant frena or muscle attachments.
3. To deepen the vestibule.
4. To repair gingival clefts.

The initial wound healing studies demonstrated the effectiveness of the technique in the treatment of the first three aforementioned problems. These same studies, however, indicated that the free gingival graft would repair narrow clefts but would not adequately bridge deep, wide gingival clefts. It has been suggested by some clinicians that the free gingival graft will repair most gingival clefts regardless of the size of the defect. Only time will tell whether this clinical impression is fact or fallacy.

Technique

1. If there is a collar of attached gingiva present, make an incision with a No. 15 blade at the mucogingival junction and preserve the collar of gingiva (fig. 16-15a). If no gingival tissue exists or the surgical objective is to cover denuded root surfaces, then dissect a partial-thickness flap at the beginning margin (fig. 16-15b).

2. Dissect the tissue close to the bone, leaving a connective tissue bed attached to bone.

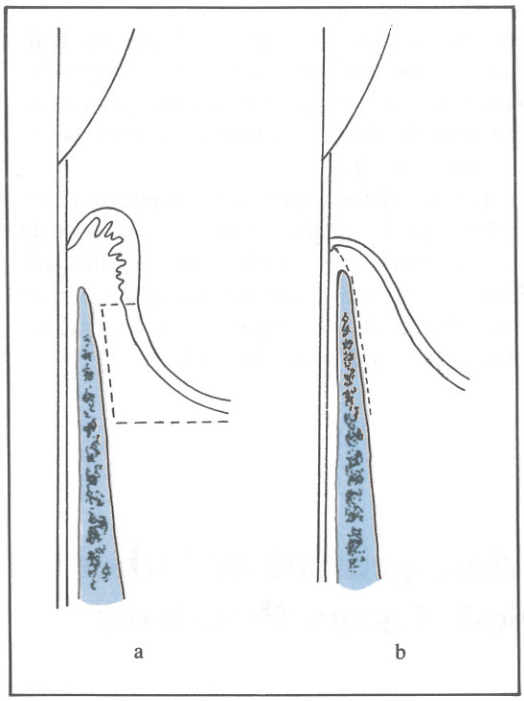

Figure 16-15

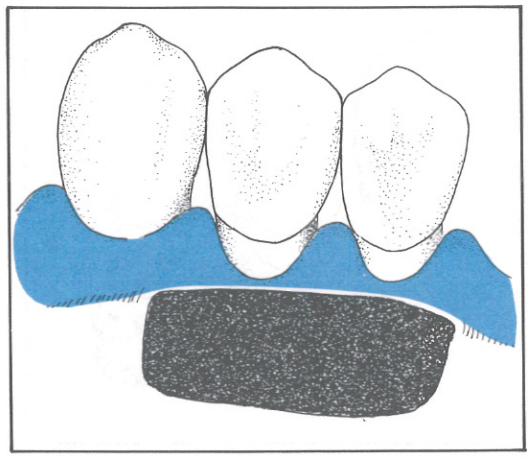

Figure 16-16

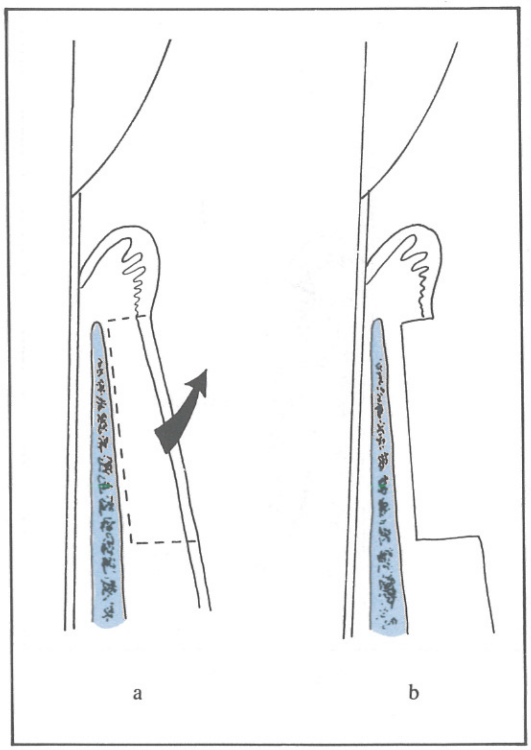

Figure 16-17

Extend the incision to include the involved teeth (fig. 16-16). Prepare the bed by removing excess connective tissue with iris scissors or tissue nippers. All muscle fibers must be removed (figs. 16-17a and 17b).

3. Prepare a template of the recipient site using the sterile surgical blade wrapper.

4. Take the template to the donor site (edentulous ridge or hard palate) and outline with your knife. If the palate is used, avoid incorporating rugae in the graft.

5. Remove the donor tissue with a No. 15 blade in an ASR handle to utilize one of the special instruments designed to remove thin sections of tissue. Keep the graft as thin as possible without perforating the tissue. This will usually result in a graft between 1.0 and 1.5 mm in thickness. The thinner the graft, the longer it will survive without circulation. This is important in achieving successful repair.

6. If the donor tissue is removed from the palate, all glandular tissue must be removed with iris scissors, since it will act as a barrier to the development of new circulation.

7. Rinse the undersurface of the graft and the recipient site with normal saline solution to remove clots. Clot formation will prevent initial nutrition of the graft by diffusion and will result in death of the graft before revascularization can occur.

8. Suture the graft at the coronal margin to ensure immobilization (fig. 16-18). Lateral sutures are usually unnecessary.

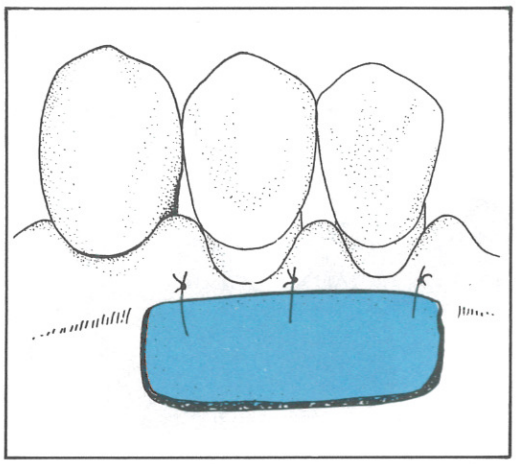

Figure 16-18

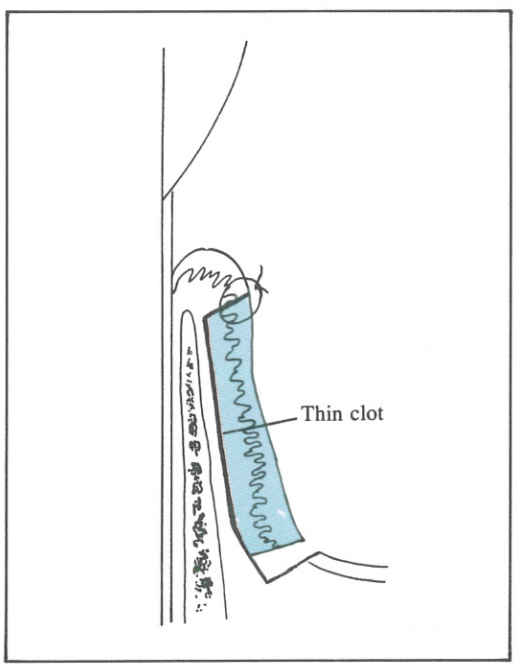

Figure 16-19

9. Apply gentle but firm pressure for 2 to 3 minutes with gauze moistened with saline solution to assist in initial fibrin clot formation and effective union between the graft and the recipient site (fig. 16-19).

10. Apply Stomahesive and cover with periodontal dressing.

11. Remove the sutures in 5 days and replace the Stomahesive and dressing for another 8 days.

12. When the final dressing is removed, caution the patient against disturbing the graft until clinical healing is complete. Advise the patient to perform plaque control procedures very carefully during this time so as not to disturb the healing graft.

It is not uncommon to see a progressive coronal shift of the gingival tissue once an adequate band of attached gingiva has been established. This phenomenon is called creeping attachment and often results in complete or at least partial coverage of an exposed root surface.

Management of Other Soft Tissue Problems

ABERRANT FRENA (FRENECTOMY)

Aberrant frena can be treated by incising the frenum at its insertion and/or grafting gingiva in its place (fig. 16-20a and 20b). Occasionally, a frenum, especially a maxillary labial or mandibular lingual frenum, is so large that it should be totally excised and the wound sutured. This is termed a frenectomy. The technique for frenectomy is as follows:

1. Grasp the frenum with a slightly curved hemostat at its base and cut the tissue with scissors above the hemostat and then below it until the hemostat is free (figs. 16-21a and 21b).

2. Use the scissors to remove any dense fibers that may be observed in the wound (fig. 16-21c). Extend the lip and check for pull on the periosteum.

3. Suture the edges of the diamond-shaped wound together (fig. 16-21d) to reduce postoperative discomfort and to promote healing.

4. Remove sutures in 7 days.

SHALLOW VESTIBULE (VESTIBULOPLASTY)

A buccal or a labial vestibule that is too shallow for placement of a removable prosthetic appliance may be deepened by a free gingival graft or an apically positioned flap (figs. 16-22a and 22b).

Figure 16-20a

Figure 16-20b

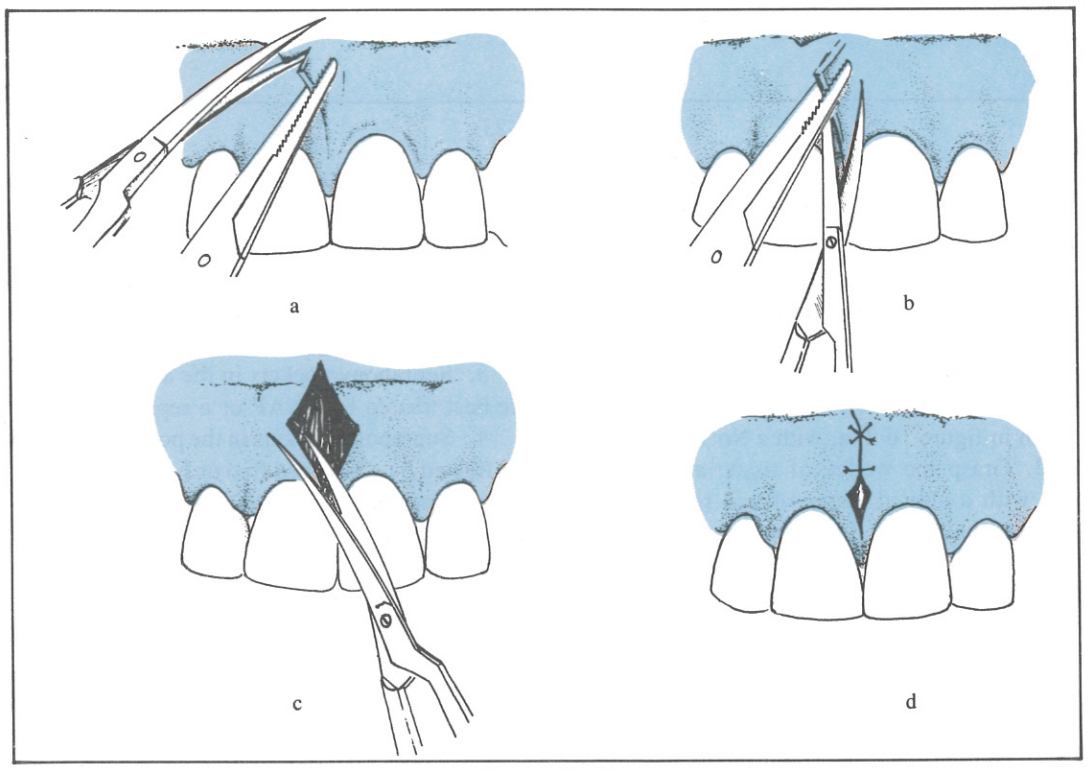

Figure 16-21

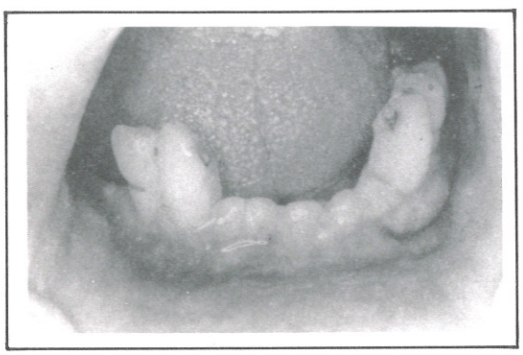

Figure 16-22a

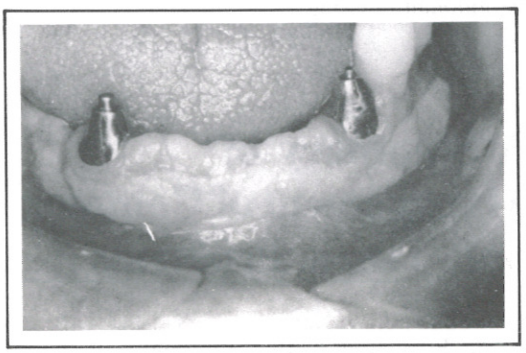

Figure 16-22b

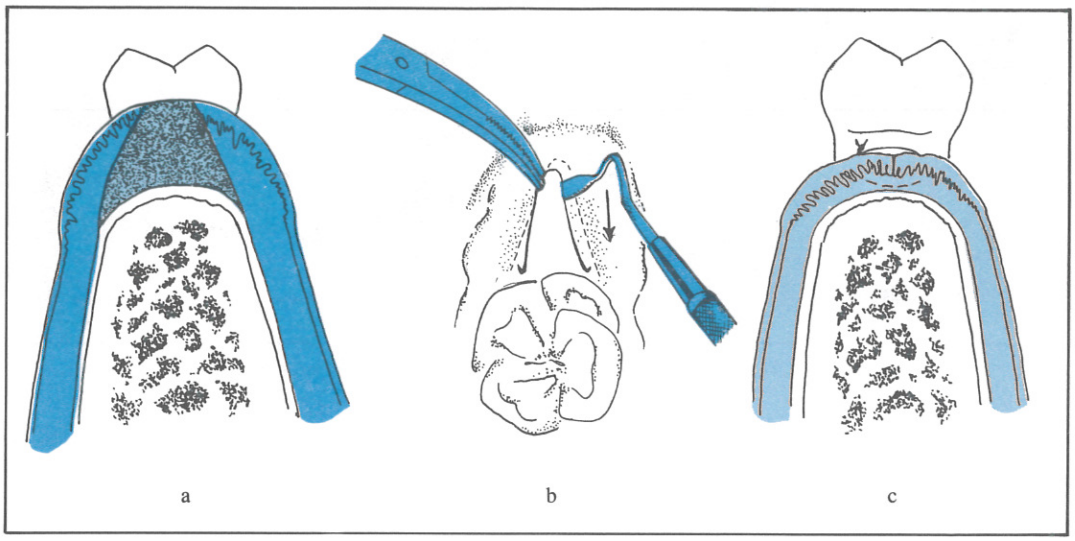

Figure 16-23

DEEP POCKET RETROMOLAR AREA (DISTAL WEDGE PROCEDURE)

A deep pocket in the retromolar area may be corrected by a distal wedge procedure. The technique is as follows:

1. Prepare a partial-thickness flap on the buccal and lingual of the retromolar area, as shown in figure 16-23a, with a No. 12-B blade.
2. Grasp the wedge of tissue at the distal edge with a curved hemostat and remove with an Orban knife (fig. 16-23b).
3. Scale and root plane the distal surface of the molar.
4. Perform osseous surgery, if indicated. This is a common area for a deep osseous defect which responds well to bone grafting procedures.
5. Approximate the wound edges and suture with interrupted sutures (fig. 16-23c).
6. Protect this area for 7 days with a periodontal dressing.

A Summary of Management of Common Soft Tissue Problems

1. Edematous, inflamed gingiva with no apical migration of the junctional epithelium can usually be remedied by scaling, polishing, and plaque control.
2. Fibrotic gingival pockets can best be eliminated by gingivectomy/gingivoplasty if adequate attached gingiva is present.
3. Suprabony pockets in the anterior region are best treated by ENAP or a replaced flap.
4. Suprabony pockets in the posterior region are treated by a replaced flap or ENAP if there is an adequate band of keratinized gingiva or by apically positioned flaps if there is an inadequate band of gingiva.
5. An adequate band of keratinized gingiva can be established by a free graft, a laterally positioned flap, a double papillae flap, or an apically positioned flap.

17 Management of osseous defects: osteoplasty/ ostectomy

Introduction to osseous surgery
- Objectives
- Diagnosis
- Classification
  - Three-wall defect
  - Two-wall defect
  - One-wall defect
  - Combination

Management
- Osteoplasty/ostectomy
  - Indications
  - Technique
  - Precautions

Introduction to Osseous Surgery

An osseous defect is a concavity or deformity in the alveolar bone involving one or more teeth. *Osseous surgery* is the general term for all procedures designed to modify and reshape defects and deformities in the bone surrounding the teeth.

OBJECTIVES

There are three basic objectives to be accomplished by osseous surgery: (1) To create contours that permit patients to accomplish effective plaque control, (2) to induce regeneration of lost supporting structures of the periodontium, and (3) to permit primary wound closure.

A rational approach to osseous surgery must be based on the accurate diagnosis and morphological classification of existing defects.

DIAGNOSIS

It is important that the therapist determine the morphology of an osseous defect as accurately as possible. It is unfortunate that most of the methods of diagnosing osseous defects record the topography in a single plane in one spatial dimension. Routine probing will supply the linear measurement of pocket depth. Radiopaque materials such as Hirschfeld points and silver points will disclose depth and contour of the pocket in respect to the bony outline (fig.

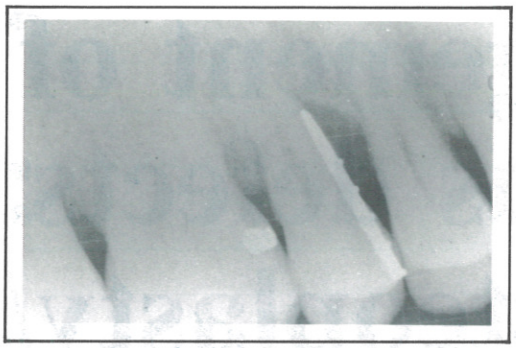

Figure 17-1

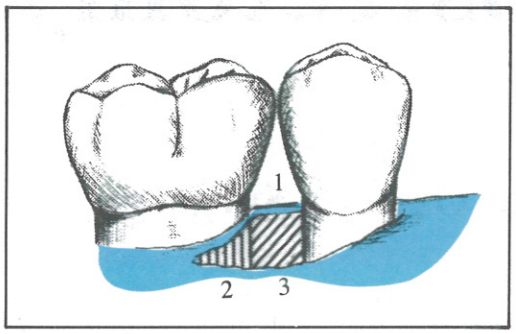

Figure 17-2

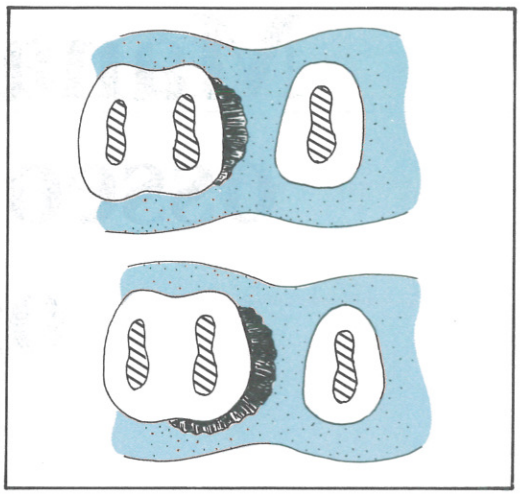

Figure 17-3

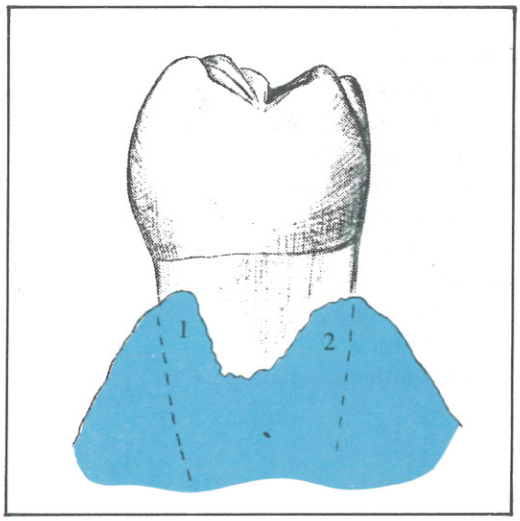

Figure 17-4

17-1). If anesthesia is used, one can probe horizontally with a sharp instrument, such as an explorer or needle, to help determine the location and the number of osseous walls. However, the three-dimensional morphology of a defect cannot usually be determined until the defect is visualized at the time of surgery.

CLASSIFICATION

Bony defects can be classified according to the number of remaining osseous walls.

Three-wall Defect

The three-wall infrabony defect occurs most frequently in the interdental region. The remaining walls are the facial, the lingual, and the proximal bone (fig. 17-2). A three-wall defect may also occur as a trough-like defect on the facial or lingual aspects. Occasionally, you will find a three-wall defect which wraps around the tooth and involves two or more contiguous root surfaces as shown in figure 17-3. In addition to designation by the number of walls, the three-wall defect is described as narrow, wide-mouth, shallow or deep, depending on its cubic dimensions.

Two-wall Defect

The most prevalent osseous defect is the two-wall crater (fig. 17-4). It is found interdentally and has a facial and a lingual wall. A two-wall defect can also occur when facial and proximal, or lingual and proximal, walls remain (fig. 17-5).

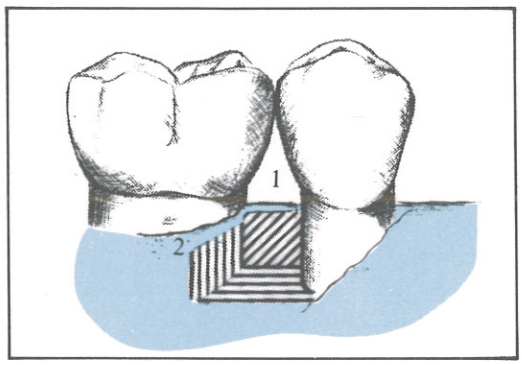

Figure 17-5

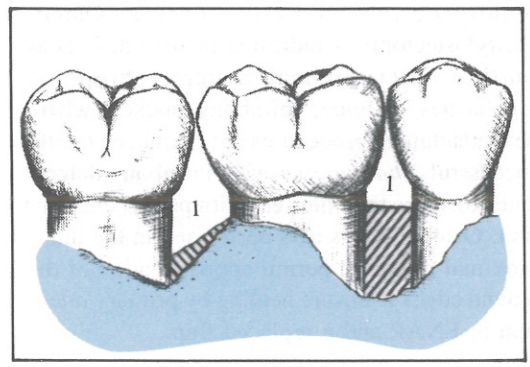

Figure 17-6

One-wall Defect

The one-wall defect usually occurs interdentally and the remaining proximal wall is sometimes referred to as a hemiseptum. The remaining osseous wall may be on the facial or the lingual side (fig. 17-6).

Combination

Many osseous lesions occur as some combinations of the one-, two-, and/or three-wall bony defect as shown in figure 17-7.

Depth, width, topography, number of remaining osseous walls, and the configuration of the adjacent root surfaces are all important in determining the therapeutic approach.

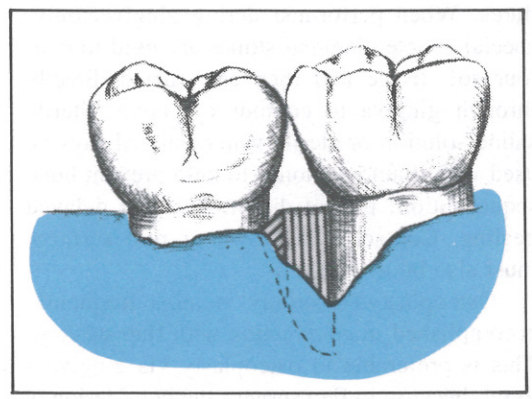

Figure 17-7

Management

The therapist has five basic choices for resolving bony defects:

1. He can eliminate the defect by recontouring (osteoplasty) or removing (ostectomy) the bone.
2. He can induce or promote regrowth and regeneration of bone. This is obviously the treatment of choice whenever possible.
3. He can amputate a root, in cases of interradicular involvement, or divide the tooth in half to eliminate the defect.
4. He can *attempt* maintenance of the pocket and osseous defect by frequent scaling, root planing, and plaque control.
5. He can extract the tooth.

These five methods of management of osseous defects will be discussed in this and subsequent chapters.

Osteoplasty/Ostectomy

Osteoplasty is the shaping of bone to restore physiologic contour. In periodontal surgery, ostectomy implies removal of alveolar bone proper and of supporting bone.

Indications

Encouraging clinical results obtained with bone grafting have somewhat modified the indications for osteoplasty/ostectomy. Also, there is evidence that some osseous defects remodel as a result of the natural healing process following flap surgery. However, until all defects are amenable to new attachment procedures or until osseous defects will remodel rapidly and predictably after flap surgery, certain defects will

require correction by contouring. Osteoplasty/ostectomy is indicated in such defects as *shallow* interproximal craters, abrupt irregularities of bone, infrabony pockets where new attachment procedures have not been totally successful, *shallow* one-wall infrabony defects and exostoses that interfere with pocket elimination. Osteoplasty is also performed in the interproximal region to permit approximation of the wound edges to ensure healing by primary intention in ENAP and a replaced flap.

Technique

Osteoplasty/ostectomy can be accomplished in conjunction with gingivectomy or flap procedures. When performed during gingivectomy, special coarse diamond stones are used to contour soft tissue and then are carried directly through gingiva to contour the bone. Sterile saline solution or sterile water must always be used with diamond stones to help prevent bone sequestration, patient discomfort, and delayed healing. Contact with crown or root surfaces must also be avoided.

Osteoplasty/ostectomy is most frequently accomplished in conjunction with flap surgery. This is preferable to osteoplasty via gingivectomy, because in flap surgery the bony lesion is visible and treatment can be determined by the topography of the defect. It is extremely difficult, and sometimes impossible, to predetermine the exact configuration and number of walls of a defect by probing and radiographic examination. If osseous surgery is indicated, chisels, surgical burs, files, rongeurs, osteotomes, and diamond stones may be used.

Precautions

Bone should rarely, if ever, be removed from radicular surfaces. Because of the thinness of bone over the root surfaces, and the absence of cancellous bone, any trauma to the bone in this area, such as that produced by a diamond stone (with or without a coolant), may result in continued bone loss during healing and formation of alveolar dehiscences.

18 Management of osseous defects: grafting

Autografts
- **Free osseous autograft**
  - **Extraoral donor sites**
  - **Intraoral donor sites**
  - **Bone blend technique**
- **Contiguous osseous autograft**

Other techniques for promoting bone regeneration
- **Flap/curettage technique**
- **Freeze-dried bone**

For many years, researchers and clinicians alike have attempted to correct osseous defects by promoting regeneration of bone with various osteogenic stimulators. Bovine bone, cartilage, plaster of paris, cementum, dentin, ceramics, freeze-dried bone allograft, fresh allograft, and the patient's own bone and marrow (autograft) have been tried, with varied success. To date autografts and freeze-dried allografts appear to offer some hope for inducing regeneration of lost supporting structures of the periodontium on a fairly predictable basis.

Autografts

Autogenous bone grafts are of two general types: free osseous autografts and contiguous osseous autografts.

FREE OSSEOUS AUTOGRAFT

Free autografts may be composed of compact, cancellous, or combined compact-cancellous bone. Clinical experience suggests that cancellous bone affords greater opportunity for "success," but it is more difficult to obtain, from the standpoint of both the patient and the therapist. Consequently, the majority of defects in which autogenous bone is used are filled with a combination of compact-cancellous bone, with the higher percentage being usually compact bone.

Autografts can be obtained from extraoral and intraoral sites.

Extraoral Donor Sites

Autogenous hemopoietic marrow/cancellous bone. This material has been used in grafting of periodontal defects for several years. It is obtained from the ilium of the patient by a cut-down procedure or by a punch biopsy technique from the anterior, superior, or posterior iliac crest. The cores of marrow and cancellous bone may be temporarily stored in Hank's solution or placed in an appropriate medium and frozen.

Studies have consistently shown that bone marrow grafts have extremely high osteogenic potential. Fresh hemopoietic marrow differs from other grafting materials in that it contains pluripotential cells which may differentiate, proliferate, and actually participate in bone formation. The basic functions of the other materials (freeze-dried bone, ceramic implants, compact and cancellous bone) are (1) to assist in bone formation by acting as a template or a trellis and (2) to induce or stimulate new bone formation. Ultimately, these materials are completely replaced, whereas bone marrow has the potential (stem cells) to form new bone without replacement. Bone marrow autografts have been used successfully in the treatment of one-, two-, or three-wall infrabony defects and some furcation involvements. Alveolar crest height has been increased in a few reported cases and, in one instance, an alveolar dehiscence has been repaired. Bone marrow has shown great promise in periodontal therapy, but it does have some disadvantages. In most instances, the services of a physician are required to obtain the material; and the experience is time-consuming, costly, and often traumatic for the patient. The bone biopsy method for obtaining the marrow is less traumatic than the cut-down procedure, but some patients express considerable apprehension about submitting to either procedure. The major problem reported with *fresh* hip marrow/cancellous bone is progressive root resorption. Some clinicians have reported extensive resorption coronal to the crestal area of the implant 6 months to 3 years after grafting. Resorption of this type has not been observed with intraoral autogenous bone or when the hip marrow/cancellous bone is frozen for delayed transplantation. The reason(s) for this resorption is (are) unknown.

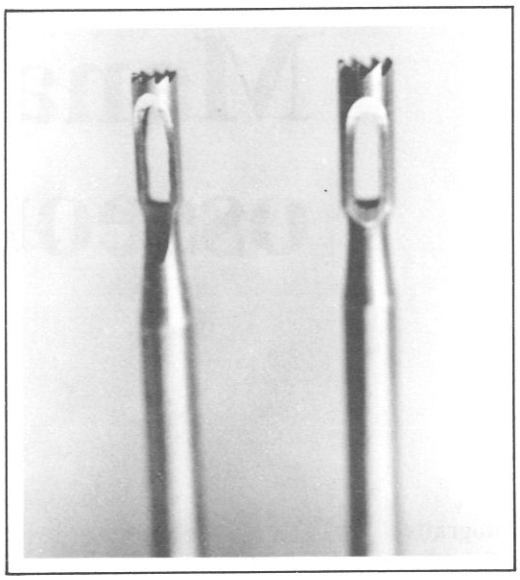

Figure 18-1

Intraoral Donor Sites

1. Bone that is removed during osteoplasty/ostectomy is an excellent source of material. The size of the chips may vary from fairly large fragments to small particles, depending on how the osteoplasty/ostectomy is performed. If rotary instruments are used, the particle size is very small. There is some evidence that small particles may more actively induce regeneration of bone in osseous defects. Small particles of bone also offer the advantage of affording a larger surface area for new bone formation. Likewise, they are more rapidly resorbed and replaced by new bone than are large bone fragments.

A technique has been described that utilizes a coagulum of blood, saliva, and small fragments of bone removed by rotary instruments to induce regeneration (osseous coagulum).

2. Bone may be obtained from a healing extraction site. About 2 months after tooth removal, a flap is made over the site, and bone is removed with end cutting rongeurs or with large curets. This is immature bone and appears to offer excellent healing and reparative potential.

3. Bone is also obtained from the tuberosity or edentulous ridges by means of rotary trephines (fig. 18-1). Care must be taken not to permit the

trephines to overheat and burn the plugs. The trephines break easily if they are stopped and then started again while engaging the plug of bone. Bone from the tuberosity area usually contains hemopoietic marrow. However, limited visual and mechanical access, together with the frequent occurrence of an alveolar extension of the maxillary sinus in the area of the tuberosity, severely reduces the availability of grafting material in this region. Some clinicians advocate removing the cortical plate of bone over the tuberosity or over an edentulous area with burs or end cutting rongeurs in order to gain access to the underlying cancellous bone.

4. A surprising amount of bone can be obtained from the operative site by utilizing chisels (Ochsenbein, Wedelstaedt), or files (Chigo, Sugarman).

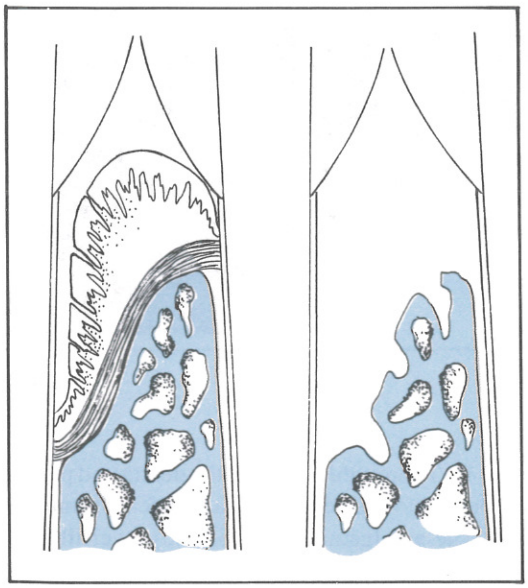

Figure 18-2

Bone Blend Technique

Intraoral autogenous bone blended in an amalgamator to a particle size of 100-300 microns is the donor material most frequently used at the Naval Graduate Dental School. This bone has been used successfully to treat one-, two-, and three-walled defects and some furcation involvements. The bone blending technique is as follows:

1. The technique for preparation of the recipient bed is the same regardless of what donor material is utilized. Make a reverse-beveled incision around the necks of the teeth to remove the inner soft tissue walls of the pocket. Preserve as much gingival tissue as possible to ensure primary closure of the wound.

2. Elevate a full-thickness or a partial-thickness flap to expose the defect. Occasionally, one or more relaxing incisions may be needed to provide better access.

3. Remove all soft tissue fibers and granulomatous tissue from within and around the defect.

4. Plane the root surface to a smooth, hard consistency.

5. Perform intramarrow penetration with a sharp instrument or a No. ½ round bur. The compact bone surrounding the defect is perforated to allow for rapid growth of new blood vessels into the area from the surrounding marrow. Intramarrow penetration also permits bone forming cells to enter the graft site (fig. 18-2).

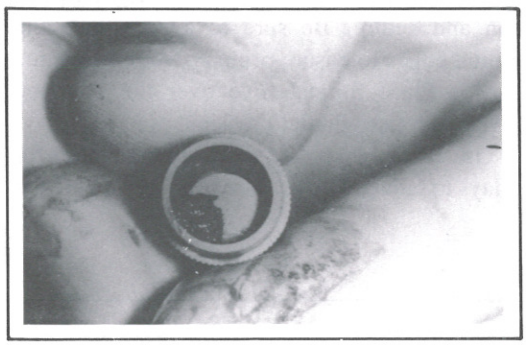

Figure 18-3

6. Obtain bone from an intraoral site as previously described (chisels, files, trephines, etc.).

7. Place the bone fragments in a new, sterile disposable amalgam capsule and pestle and add a drop of saline solution (fig. 18-3).

8. Wrap the capsule in sterile gauze; place it in the amalgamator and triturate it for 60 seconds. Hold the capsule in the amalgamator during trituration to prevent it from slipping from the amalgamator. A dense mass of bone may require more time for blending.

9. Observe the bone blend clinging to the walls of the capsule and to the pestle (fig. 18-4). Remove the blend with a spoon-shaped instrument.

10. Transfer the slushy mix of bone particles to a sterile dappen dish.

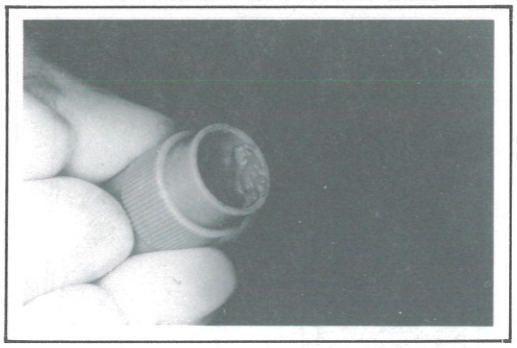

Figure 18-4

11. Thoroughly aspirate the defect and place the bone into the defect with a plastic instrument or sterile amalgam carrier. Slightly overfill the defect with bone to the crestal height. The bone will quickly become mixed with blood from the recipient site.

12. Replace the flap(s) in its original position and suture. Be sure to approximate the wound edges to ensure primary wound closure.

13. Place Stomahesive over the flap and cover with periodontal dressing.

14. Prescribe broad spectrum antibiotics for 7 days to reduce plaque formation. Also give the patient an analgesic to control any postoperative pain.

15. Apply an ice pack immediately over the region of the operation to reduce swelling. Encourage the patient to reapply ice three to four times thereafter.

16. Remove sutures in 7 to 10 days and redress the area for another week.

17. When the dressing is removed, instruct the patient in plaque control. Do not probe the graft site for 3 months.

CONTIGUOUS OSSEOUS AUTOGRAFT

In this type of graft, bone adjacent to a defect is partially fractured from its base with a chisel and then wedged into the defect against the root surface (figs. 18-5a, b). The technique has been likened to a greenstick fracture. It has been used to eliminate the osseous defect (one-wall) that is so often associated with a tilted molar (figs. 18-5c, d). However, there is some evidence that infrabony defects of this type can be eliminated, or at least reduced in dimension, by orthodontically uprighting tipped molars. Consequently, it may be preferable to upright teeth prior to performing grafting procedures.

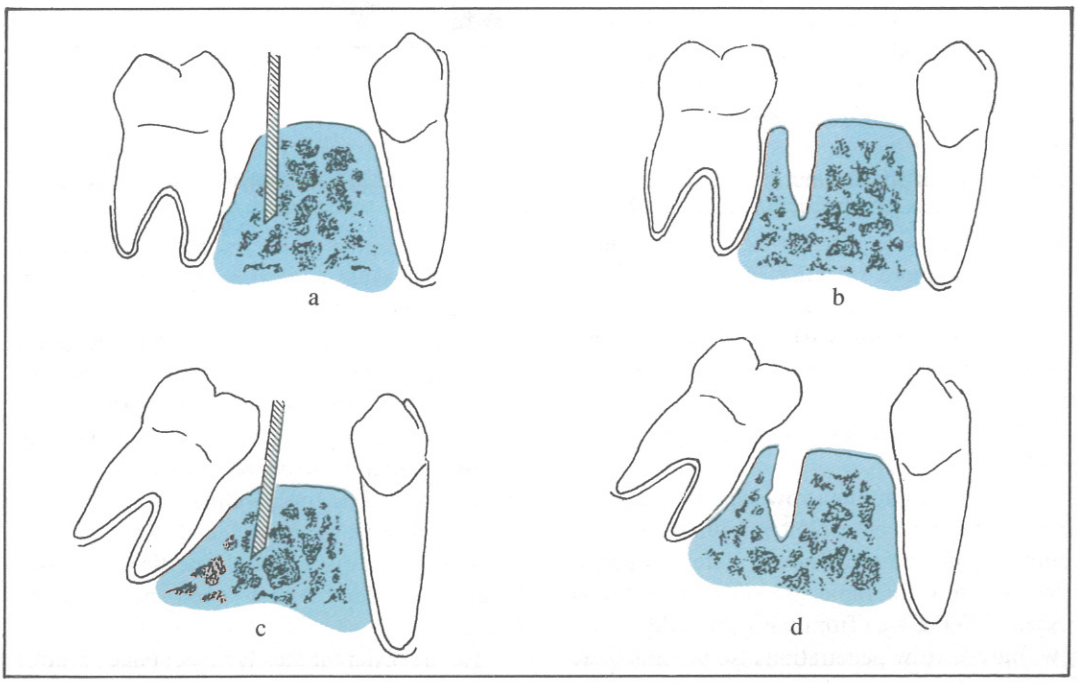

Figure 18-5

Other Techniques for Promoting Bone Regeneration

FLAP/CURETTAGE TECHNIQUE

This procedure is performed exactly as that described for other grafting techniques except that donor bone is not utilized. It is advocated by some clinicians as the treatment of choice for obtaining new attachment in narrow three-wall defects.

FREEZE-DRIED BONE

Freeze-dried bone is an osseous allograft that is obtained from a cadaver, freeze-dried, pulverized to a small particle size (100-300 microns), placed in a vacuum bottle, and stored. Freeze-dried bone has been used successfully for over 20 years in medicine and dentistry. Recent studies indicate that this material offers promise in the management of osseous defects of the periodontium. It has also been used in combination with autogenous bone to supplement the small amount of autograft that can be obtained intraorally. Clinical impression suggests that when freeze-dried allograft and autograft are mixed together, there is even greater osteogenic potential than when either material is used by itself. Further studies are being conducted to evaluate this observation.

19 Management of osseous defects: furcation involvements

Treatment considerations
- Philosophy
- Diagnosis
- Classification
- Prognosis
- Pulpal-periodontal relationships

Therapy
- Class I involvement
- Class II involvement
- Class III involvement
  - Bone grafting
  - Enlargement
  - Root resection
  - Hemisection

Treatment Considerations

PHILOSOPHY

Treatment of teeth with furcation involvements is a complicating factor in periodontal therapy. A furcation involvement may be defined as a pathologic condition that has destroyed the periodontium in the interradicular area of a multirooted tooth. Opinions differ as to whether these teeth should be treated or extracted. Recently, the trend has been toward retaining such teeth, especially if they are of strategic importance in the overall treatment plan. While furcation pathology is still a serious challenge, advances in treatment procedures have increased the number of teeth with such involvement that can be preserved. Nevertheless, the dentist is cautioned to avoid unnecessary heroics in attempting to salvage seriously involved multirooted teeth by means of "interesting" techniques. Before attempting extensive therapy, he should ask the following questions:

1. Will retention of this tooth preserve arch integrity and obviate prosthetic replacement?
2. Will its retention permit better prosthetic design?
3. Is the tooth vital to an existing prosthesis?
4. Can the proposed therapeutic effort be classified as bona fide therapy, or is it merely an academic exercise?

DIAGNOSIS

Furcation pathology is diagnosed by use of a periodontal probe, a pigtail or cowhorn explorer, and radiographs. The radiograph may reveal evidence of a furcation involvement; whereas probing reveals that the soft tissue attachment is still intact, with no entrance into the furcation area. Obviously, the clinical examination is the critical test in this instance.

Maxillary molars with extensive pocket depth (5 mm or more) on the mesial or distal aspect should automatically be suspected of having furcation involvement. In mandibular molars, extensive midbuccal or midlingual pocket formation strongly suggests interradicular pathology, regardless of the radiographic evidence.

Probing and positive identification of maxillary furcation involvement can be especially difficult. Occasionally, anesthesia must be employed to permit adequate diagnosis. Mesial and distal entries to the furca are best established from the palatal aspect. Although careful presurgical diagnosis can minimize the possibility of a ''surprise'' furcation problem, it is not uncommon to find final, positive evidence only at the time of surgery.

CLASSIFICATION

Most classification systems are based on a division of furcation pathology into three classes: Class I (incipient defects), Class II (moderate involvement), and Class III (through-and-through communication). These classes may be defined in greater detail as follows:

Class I: A soft tissue lesion extending to the furcation level but with minimal osseous destruction. Radiographs of these incipient lesions reveal little, if any, evidence of pathology (fig. 19-1).

Class II: A soft tissue lesion combined with bone loss that permits a probe to enter the furcation from one aspect but not to pass completely through the furcation. This class is further subdivided as follows (fig. 19-2):

Class II-F: Entry is from the facial aspect only.

Class II-L: Entry is from the lingual aspect only.

Class II-M: Entry is from the mesial aspect only.

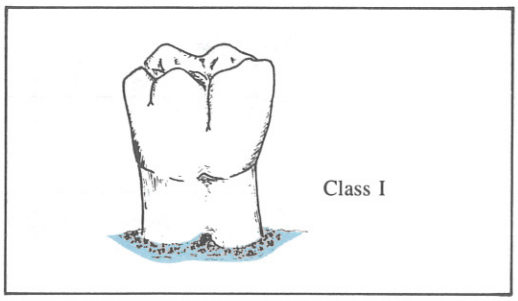

Figure 19-1

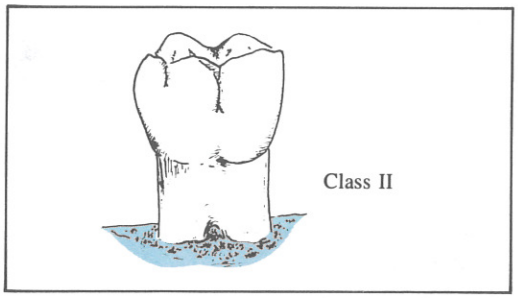

Figure 19-2

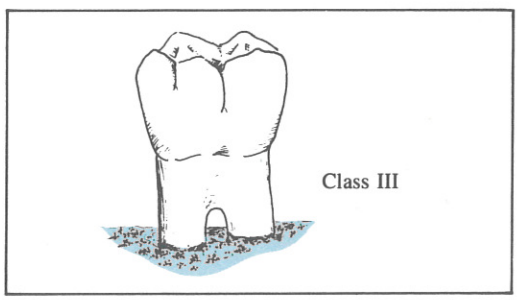

Figure 19-3

Class II-D: Entry is from the distal aspect only.

Some evidence of Class II involvement can usually be observed in radiographs of mandibular molars, but such evidence may be obscured in radiographs of maxillary molars.

Class III: Lesions with extensive osseous destruction that permit through-and-through passage of the probe. The through-and-through nature of the communication may be obscured by soft tissue. Some classifications include a fourth category (Class IV) when there is complete visualization through the furcation (fig. 19-3).

PROGNOSIS

The prognosis of a furcation involvement depends on the following:

1. Extent of horizontal and vertical bone destruction in the interradicular space.
2. Number of roots and their morphology.
3. Morphology of the interradicular space (width, depth, etc.).
4. Health status of the periodontal ligament (as determined by tooth mobility, percussive response, etc.).
5. Access for surgical correction.
6. Access for plaque control by the patient following surgical correction.
7. Pulpal status and prospects for successful endodontic therapy and root removal procedures.
8. Possibility of controlling occlusal factors.

When the foregoing factors are equal, mandibular first molars generally have the best prognosis, followed by mandibular second molars and maxillary first molars. The prognosis of maxillary bicuspids is considered poor, even when there is only moderate furcation pathology. Anatomically, these teeth do not lend themselves to satisfactory plaque control or root amputation. Likewise, the possibility of reattachment in the involved furcation areas of a bicuspid is poor. Freeze-dried bone allograft, plus autograft donor material, or frozen marrow autograft, may offer future hope for such teeth.

PULPAL-PERIODONTAL RELATIONSHIPS

The relationship between pulpal disease and periodontal disease has been increasingly appreciated. It is apparent that there are many direct communications between pulpal tissue and the periodontal ligament. Dentists can no longer afford the "security" of considering these areas as separate environments. In diagnostic terms, this means that a complete pulpal evaluation should be a part of every periodontal examination.

Pulpal-periodontal interaction is especially important in interradicular areas. Because of the many accessory foramina in the furcation areas, Class II and Class III furcation lesions are sometimes associated with pulpal disease. It has been demonstrated that the interradicular periodontal apparatus is especially sensitive to excessive occlusal stress. Thus, the combination of pulpal disease, periodontal traumatism, and inflammation might reasonably be expected to produce extensive destruction in the furcation area. (This etiologic relationship is yet to be confirmed by research.)

The many uncertainties in pulpal-periodontal relationships provide continual diagnostic challenges to the therapist: Is the periodontal lesion primarily an extension of pulpal disease? Or, has periodontal inflammation interfered with pulpal health? To compound this dilemma, both areas may be in deteriorating states that will ultimately demand both periodontal and endodontic therapy but, in the interim, will severely test the diagnostician's skills.

Consequently, the modern therapist must be highly suspicious of pulpal disease, especially when the following conditions exist:

1. Periodontal pockets near or leading to the furcation area or apex.
2. Fistulae of uncertain origin.
3. Discolored teeth.
4. Chronic drainage from the sulcus.
5. History of acute or chronic pulpal insult (periodontal traumatism, extensive restorative dentistry, etc.).
6. Prolonged hypersensitivity.
7. Evidence of slow or inadequate healing of periodontal lesions.

Therapy

CLASS I INVOLVEMENT

Treatment for incipient furcation lesions is essentially the same as for an uncomplicated soft tissue pocket. If the width of attached gingiva is adequate, gingivectomy/gingivoplasty is often employed, together with thorough debridement and root planing. This is accomplished to permit access to the furcation for both therapist and patient.

1. Careful attention should be given to the character of the cervical crown area. Inadequate Class V restorations, cervical caries, and generally poor crown contours may be etiologic factors which should be corrected.

2. Projections of enamel into the furcation area may influence the spread of gingival inflammation. This anomaly occurs on the buccal

aspect of about 25 percent of all molars. Although the importance of such projections is not clear, the therapist should be aware of them. Removal of the enamel projections by odontoplasty may be indicated, especially if reattachment is anticipated. On the other hand, if the furcation is to be left permanently exposed, removal of these anomalies may cause unnecessary dentinal sensitivity.

CLASS II INVOLVEMENT

In situations where there is major destruction of furcation integrity—but short of through-and-through penetration—various flap procedures are necessary for adequate access and instrumentation. If there is an adequate width of keratinized gingiva, replaced flaps can be utilized to attempt new attachment into the furca. Perhaps the most predictable therapy is to apically position the flap on the facial or lingual surface and permit the patient to cleanse into the furca. Deliberate debridement and root planing, as well as careful flap stabilization, are important in treating these defects. In complicated furcation anatomy, finishing burs can be used as an effective supplement to conventional root planing instruments.

Occasionally, interfurcal (interradicular) osseous craters will be observed. This type of defect is particularly amenable to correction by bone grafting techniques.

CLASS III INVOLVEMENT

There are several categories of therapy for Class III furcation involvement:

1. Completely reestablish furcation integrity by osseous grafting techniques.
2. Increase the furcation opening to facilitate plaque control.
3. Eliminate the furcation by various root removal procedures.
4. Extract the tooth.

Bone Grafting

In the past, attempts to reestablish total furcation integrity—bone, periodontal attachment apparatus, and dentogingival relationship—were usually unsuccessful. This goal was considered practically unattainable. Today, bone grafting techniques offer some promise of success in achieving this most difficult periodontal objective. This is particularly true if interfurcal osseous craters are present.

Enlargement

Enlargement of an existing Class III furcation defect may facilitate plaque control and permit retention of the tooth. Enlargement may be at the expense of either tooth structure or bone. This approach, however, is generally limited to mandibular molars. Occasionally, a trifurcation area can be opened widely, but adequate plaque removal is difficult. Even after apparently successful treatment of Class III furcation involvement, caries in the furcation area is a constant threat. Patients with through-and-through furcation involvement should receive topical fluoride application periodically and are advised to use a fluoride dentifrice.

Root Resection

Root amputation is the most predictable procedure for Class III trifurcation involvement. It is also indicated in bifurcations when new attachment procedures are contraindicated or are unsuccessful. The root with the greatest overall bone loss is the logical candidate for amputation. If there is no marked difference, the mesial root is generally removed, since plaque control is more easily accomplished on the mesial surface of the remaining roots. There are several considerations prior to root resection.

Considerations.

1. The therapist should study root and furcation anatomy on extracted molars prior to surgery.
2. Occlusion should be checked and adjusted. The occlusal table should be narrowed, and balancing and centric prematurities should be removed (fig. 19-4).
3. The need for splinting should be established preoperatively.
4. The strategic importance of teeth indicated for root resection should be thoroughly evaluated.
5. The maxillary sinus should be respected and avoided.

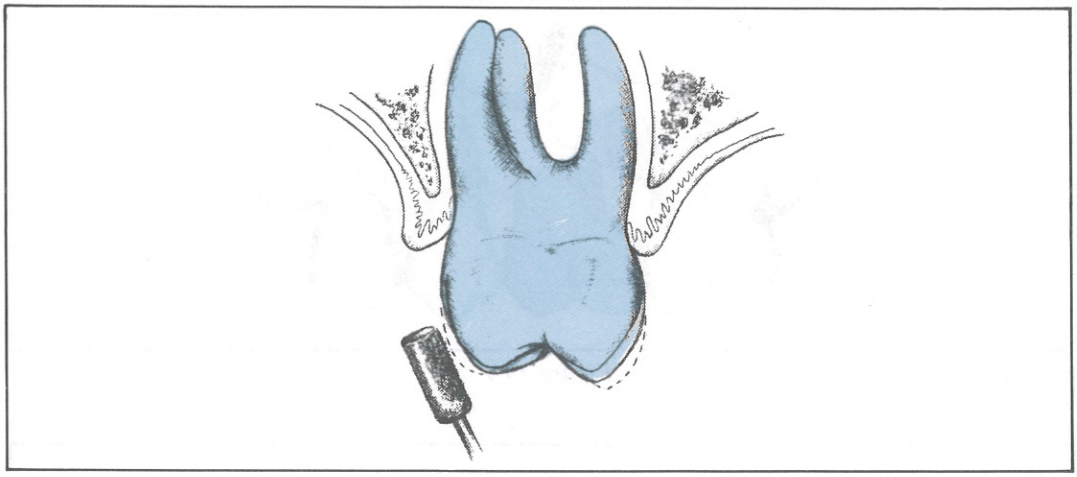

Figure 19-4

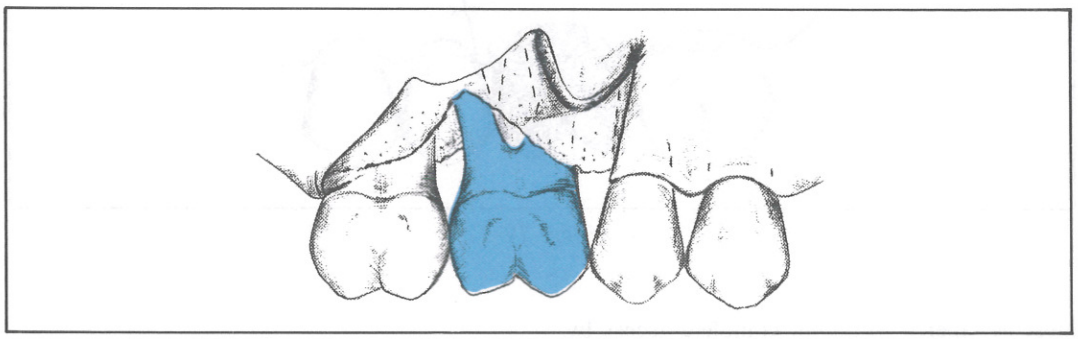

Figure 19-5

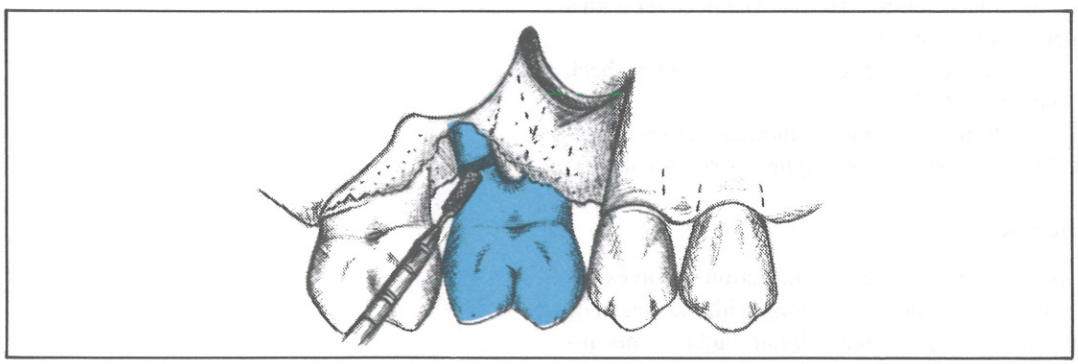

Figure 19-6

Technique for root resection.

1. Accomplish endodontic therapy prior to root amputation.

2. Elevate a mucoperiosteal flap to expose the defect (fig. 19-5). One and sometimes two vertical incisions are required for adequate access.

3. Make the initial cut with a cross-cut bur apical to the cementoenamel junction. Amputation should be at the expense of the root rather than of the crown (fig. 19-6).

4. Remove the root with end cutting rongeurs or anterior forceps.

5. Contour the resected root stump with a diamond point or fine stone. The root surface

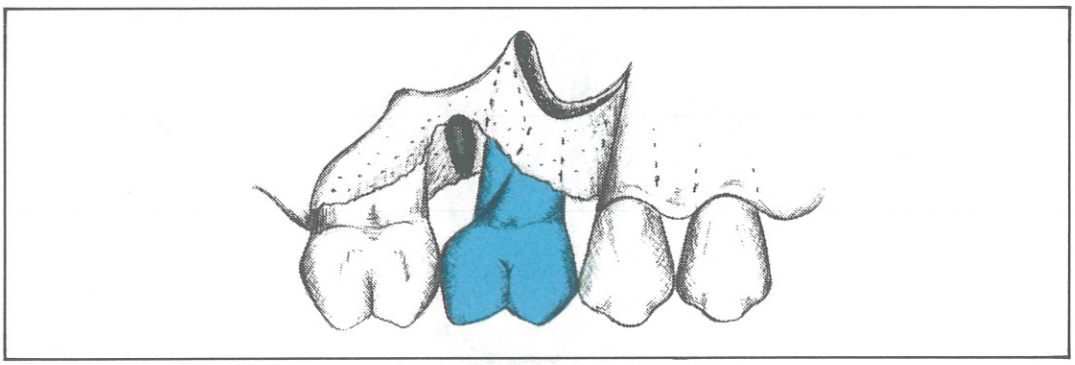

Figure 19-7

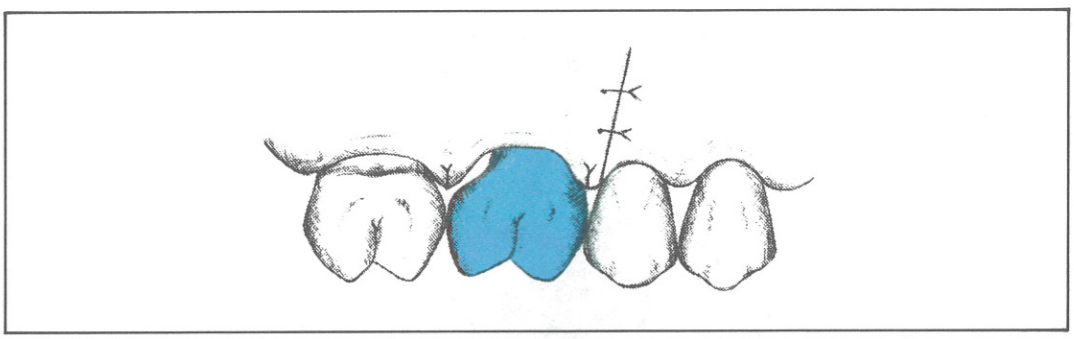

Figure 19-8

must be tapered to permit complete access by patient and also to permit passage of food (fig. 19-7).

6. Place sutures (fig. 19-8) and cover with a periodontal dressing.

7. Remove sutures in 1 week and recheck contour.

8. Polish stump with fluoride pumice. Topical fluoride should be applied periodontally.

Hemisection

As the name implies, hemisection involves removal of half the crown along with the mesial or distal root of the mandibular molar. This involves the same technique as described for root resection. The retained root can often serve as a suitable abutment for fixed splinting.

20 Management of osseous defects: additional techniques

Scaling, root planing, and plaque control
  Indications
Extraction
  Selective extraction
Treatment of osseous defects summarized

Scaling, Root Planing, and Plaque Control

On occasion, it is good therapy to manage surgically untreatable pockets and osseous defects by scaling, root planing, and plaque control rather than by extraction. It is unlikely that such pockets are ever successfully "managed" by scaling and root planing. It is more likely that the frequent removal of subgingival plaque and calculus simply retards the rate of progression of periodontal disease. If this therapy is to be of any value, scaling and root planing must be accomplished at frequent intervals (every 2 to 3 months); and in the interim, the patient must accomplish meticulous plaque control. Periodic scaling and root planing of deep pockets is considered by some clinicians to be temporizing. In any case, the advantages of retaining teeth with deep pockets must be carefully weighed against the possible disadvantages, such as potential abscess formation or loss of adjacent bone from sound teeth.

INDICATIONS

Ideally, pockets should be eliminated to permit patients to accomplish effective plaque control and to *arrest* the disease process. If this is not possible, for whatever reason, management by scaling, root planing, and plaque control should be *considered*. Some of the more common condi-

tions where this therapy can be utilized are as follows:

1. A critical but hopelessly involved tooth that is an abutment for a fixed or removable partial denture.
2. Extensive bone loss around a single tooth in an intact arch, where retention does not jeopardize adjacent teeth.
3. Extensive generalized bone loss in an elderly patient.
4. A patient who is a poor surgical risk, e.g., the alcoholic or acute leukemic.
5. A patient with extreme fear of periodontal surgery.
6. A patient with periodontal disease who will not agree to periodontal surgery or extraction.
7. A patient who does not or cannot accomplish effective plaque control, but for whom extractions are not indicated.

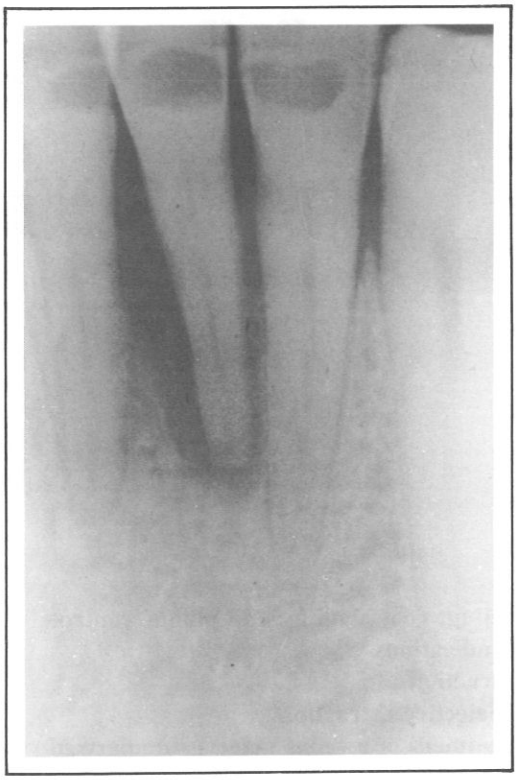

Figure 20-1

Extraction

There are those who believe the only predictable method of treating periodontal disease is extraction of the involved teeth. This philosophy can be likened to that of the physician who believes the only predictable treatment for hangnail is amputation of the finger, since hangnails are prone to recur. Extraction represents the ultimate failure in dentistry; yet, on occasion it can be used to advantage.

SELECTIVE EXTRACTION

Selective extraction (strategic extraction) of some periodontally involved teeth can significantly improve the prognosis of adjacent teeth. For example, in figure 20-1, severe periodontal involvement of this vital right central incisor has also resulted in bone loss of the adjacent teeth. The tooth exhibits Class III mobility and its prognosis is poor. If it is extracted, the socket will fill approximately to the highest level of alveolar crest on the adjacent teeth, as shown in figure 20-2. Consequently, the extraction of one tooth with a poor prognosis results in an improved prognosis of two adjacent teeth (fig. 20-3). Selective extraction is a consistent and predictable method of case management. It is a technique to be used but not abused.

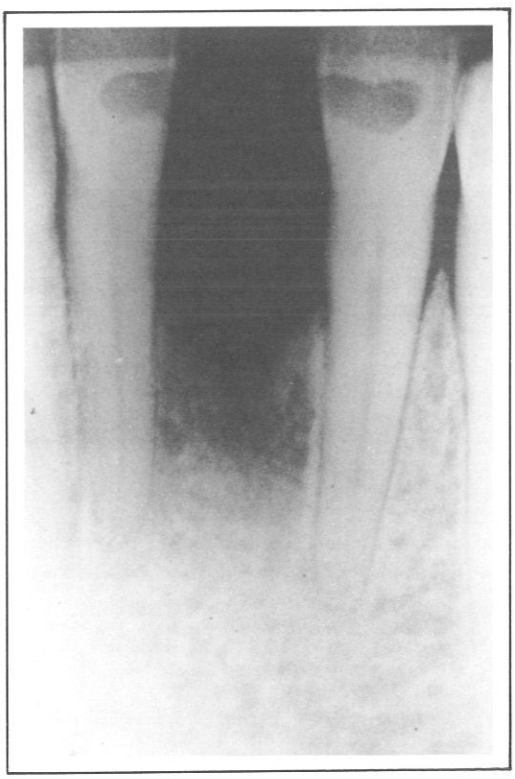

Figure 20-2

Figure 20-3

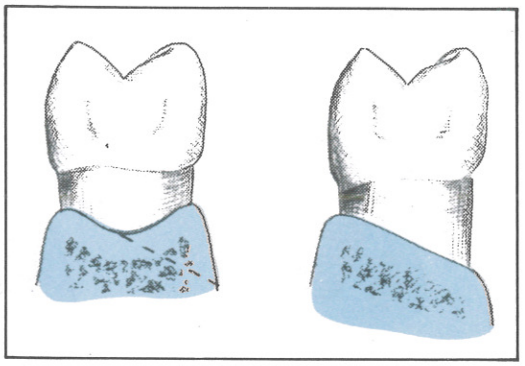

Figure 20-4

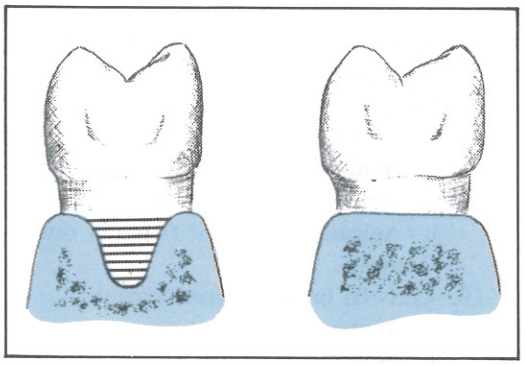

Figure 20-5

Treatment of Osseous Defects Summarized

Prognosis of infrabony pockets is influenced by the number of remaining walls, size of the defect, number of root surfaces involved, extent of bony destruction, and furcation involvement. As a general rule, the greater the number of osseous walls and the narrower the defect, the better is the prognosis for new attachment, provided there is no furcation involvement. Following is a summary of therapy for various osseous defects:

1. Broad interproximal ledges (lipping)—osteoplasty.
2. Abrupt interproximal irregularities of bone—osteoplasty.
3. Exostoses that interfere with pocket elimination—osteoplasty.
4. Three-wall defect:
   a. Narrow: (1) Bone blend technique.
      (2) Flap-curettage technique.
   b. Broad (widemouthed): Bone blend technique or freeze-dried bone.
5. Two-wall defect:
   a. Shallow crater: Osteoplasty/-ostectomy (fig. 20-4).
   b. Deep crater:(1) Bone blend technique (fig. 20-5).
      (2) Freeze-dried bone.

A second stage procedure is sometimes necessary after 12 months or more. If possible, the defect is grafted again. If it is too shallow for grafting, perform ostectomy (fig. 20-6).

6. One-wall defect:
   a. Shallow: Osteoplasty/ostectomy, if adjacent tooth is not jeopardized. Otherwise, use bone blend technique.
   b. Deep: (1) Bone blend technique.
      (2) Freeze-dried bone.
      (3) Contiguous osseous autograft, if defect is adjacent to edentulous area.
7. Combination defect:
   a. Bone blend technique.
   b. Freeze-dried bone.

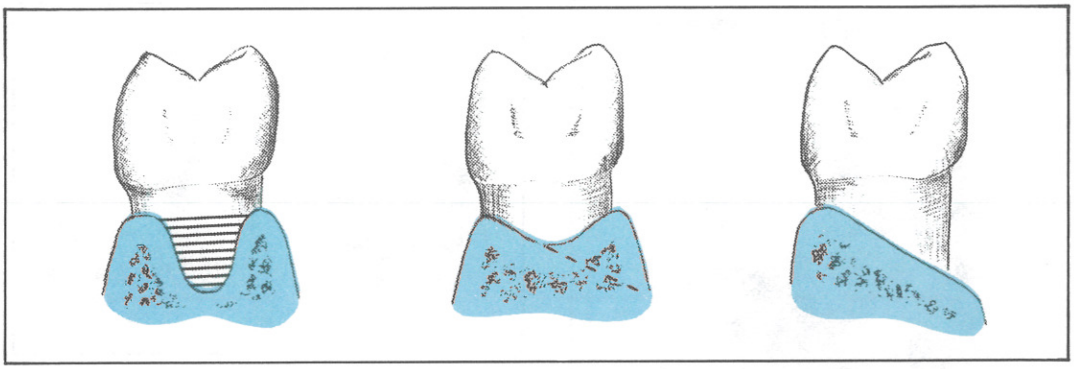

Figure 20-6

8. Class I furcation involvement—gingivectomy/gingivoplasty.
9. Class II furcation involvement:
   a. Frozen marrow graft.
   b. Bone blend technique—if interfurcal crater is present.
   c. Apically positioned flap and osteoplasty.
10. Class III furcation involvement:
   a. Root resection.
   b. Frozen marrow graft.
   c. Open defect for cleansing—through-and-through.
   d. Hemisection.

21 Periodontal emergencies

Pericoronitis
- Etiology
- Signs and symptoms
- Treatment

Abscess formation
- Gingival abscess
- Periodontal abscess
- Acute periapical abscess

Chemical and physical injuries

Necrotizing ulcerative gingivitis
- History
- Incidence
- Contagion
- Etiology
- Diagnosis
- Differential diagnosis
- Treatment of necrotizing ulcerative gingivitis

Hypersensitivity
- Etiology
- Treatment

Primary periodontal traumatism
- Definition
- Signs and symptoms
- Treatment

Temporomandibular joint pain dysfunction syndrome
- Etiology
- Symptoms
- Treatment

A periodontal emergency is any circumstance, or combination of circumstances, that adversely affects the periodontium and requires immediate attention. This definition encompasses a wide variety of conditions that involve the periodontium; however, this discussion will be limited to the emergencies most commonly encountered.

Pericoronitis

ETIOLOGY

Pericoronitis is probably the most common periodontal emergency, and the partially erupted or impacted mandibular third molar is the site most frequently involved. The overlying gingival flap is an excellent harbor for the accumulation of debris and an ideal breeding ground for bacteria. Additional insult to the pericoronal flap is often produced by trauma from an opposing tooth.

SIGNS AND SYMPTOMS

The clinical picture is a red, swollen, possibly suppurating lesion that is extremely painful to touch. Swelling of the cheek at the angle of the jaw, early necrotizing ulcerative gingivitis, partial trismus, lymphadenopathy, and radiating pains to the ear are common findings. The patient may also have systemic complications, such as fever, leukocytosis, and general malaise.

TREATMENT

The treatment of pericoronitis consists of irrigation of the under surface of the flap and the surrounding area with warm saline solution. A 10 cc syringe with a blunt 18-gauge needle, bent at an 80° angle, is an excellent irrigating

instrument. An ultrasonic instrument can also be used effectively in this region. It may be necessary to extract the opposing third molar at the first visit if it impinges on the pericoronal flap. The patient is instructed to rinse with warm salt water every 2 hours, and antibiotics are administered if systemic complications are present.

Once the acute symptoms have subsided, a careful evaluation is made to determine whether the tooth should be retained.

Abscess Formation

GINGIVAL ABSCESS

A gingival abscess is a localized, painful, rapidly expanding lesion that appears suddenly in the marginal gingiva or interdental papilla. The lesion consists of a purulent focus in connective tissue. It is initiated by the forceful embedding into the gingiva or gingival sulcus of a foreign body (e.g., a toothbrush bristle or popcorn husk). A gingival abscess may occur in tissue entirely free from periodontal disease.

Treatment consists of drainage to relieve the acute symptoms and removal of the foreign body. If the lesion has become fluctuant, topical anesthesia is applied to the gingival margin. The gingival sulcus is gently opened to permit evacuation of pus and then rinsed with warm water. The patient is advised to rinse with warm salt water every 2 hours. Once the irritant is removed and drainage is established, the tissues usually return to normal with no further treatment.

PERIODONTAL ABSCESS

A periodontal abscess is a localized, purulent inflammatory process involving the deeper periodontal structures. Abscess formation is usually associated with infrabony pockets, tortuous pockets, and furcation involvements. Obviously, conditions that either prevent free drainage or occlude the orifice of a pocket may result in abscess formation.

Periodontal abscesses may be acute or chronic. Acute lesions often subside and persist in the chronic state, while chronic lesions may suddenly become acute. The clinical signs of an acute abscess are severe pain, swelling of the soft tissues, tenderness to percussion, extrusion, and mobility of the involved tooth. Periodontal destruction in an acute abscess is rapid and extensive, and treatment should be instituted promptly.

Treatment of the periodontal abscess is performed in two stages. The first stage involves management of the acute symptoms by drainage. Whenever possible, drainage is established through the lumen of the pocket. If this cannot be done, as is often the case when there is a furcation involvement or a tortuous pocket, then drainage is obtained externally by making a stab wound through the pointed lesion. The patient is advised to rinse with warm salt water every 2 hours and is placed on antibiotics if systemic complications are evidenced. It is often necessary to adjust the occlusion of the involved tooth or teeth.

The second phase of treatment is directed toward elimination of the pocket as soon as the acute symptoms have subsided and before the chronic stage is reached. Treatment consists of gentle elevation of a mucoperiosteal flap. All granulomatous tissue is removed and the root surface is planed. Emphasis is placed on gentle manipulation of the soft tissue. The flap is replaced in its original position and sutured. A periodontal dressing is used for 7 to 10 days. Clinical experience has demonstrated a marked propensity for healing and repair following an acute periodontal abscess. This is sometimes true even when there has been marked periodontal destruction. For this reason, teeth involved by an acute periodontal abscess should be carefully evaluated before extraction is recommended.

ACUTE PERIAPICAL ABSCESS

It is sometimes necessary to differentiate between a periodontal and a periapical abscess. A nonvital pulp is usually indicative of a periapical abscess, and the tooth should be treated endodontically or extracted. A clinically vital pulp, however, is not always assurance that the problem is still not pulpal. Radiographs are of some assistance in differential diagnosis, but clinical findings such as extensive caries, tooth vitality, pocket formation, and continuity between the abscess and the gingival margin are of greater practical significance.

Various investigators have confirmed pulpal pathosis and infection in periodontally involved teeth. Thus, the probability exists that periodontitis can result in death of the pulp. It has also been demonstrated that, as a result of pulpal disease, tissue destruction may proceed from the apical region toward the gingival margin. This process is termed *retrograde periodontitis* in order to differentiate it from marginal periodontitis, in which the disease spreads from the gingival margin to the apex. Whether the periodontal pocket is a result of retrograde periodontitis or marginal periodontitis, or a combination of both, is academic. In all cases, treatment will consist of combined endodontic-periodontal therapy or extraction of the tooth.

Chemical and Physical Injuries

Injuries caused by toothbrush trauma, chemical burns, cheek and tongue biting, and periodontal dressings are occasionally observed. Emergencies of this type are painful but otherwise of little consequence. Healing usually occurs uneventfully in 10 days to 2 weeks. Treatment is chiefly symptomatic, and patient discomfort is controlled through the use of topical anesthetics or warm saline rinses. Some clinicians recommend the use of Orabase, or Orabase with triamcinolone, as palliative treatment for such injuries.

Necrotizing Ulcerative Gingivitis

HISTORY

Necrotizing ulcerative gingivitis (NUG) is an emergency of special importance and magnitude in the armed services. As long ago as 400 B.C., Greek soldiers were plagued by what appears to have been NUG. In the 1890's, Plaut and Vincent were the first to associate specific organisms with the disease process, hence, the term "Vincent's infection" has been associated with the disease for many years. The condition is also called Vincent's stomatitis, Vincent's disease, Plaut-Vincent's disease, Plaut-Vincent's stomatitis, trench mouth, and many other names. Since the trend in dental and medical terminology is to dispense with the use of eponyms, the descriptive term "necrotizing ulcerative gingivitis" is preferred.

INCIDENCE

An increase in NUG among servicemen after long furloughs and after living under field conditions for a week or more has been reported. NUG has also been related to tobacco, increased consumption of alcohol, low socioeconomic status, poor nutrition, age, general debilitation, and climate. In effect, any factor which *increases emotional stress, lowers patient resistance,* or *inhibits plaque control* can contribute to the initiation of NUG.

CONTAGION

For many years NUG was considered a communicable disease contracted from eating utensils, personal contact, etc. Numerous studies have failed to establish any pattern of transmission among individuals. Some hardy investigators have even injected fusospirochetal microorganisms in their own mouths and have not contracted the disease. The 1966 World Workshop in Periodontics concluded that NUG was not a communicable disease, based on existing evidence.

ETIOLOGY

The etiological factors in NUG can be divided into two groups: predisposing and causative factors.

1. *Predisposing factors.* These are both local and systemic. Local factors include calculus, gingival flaps over molar teeth, caries, overhanging margins of restorations, improper tooth contacts, malpositioned teeth, and food impaction. Systemic predisposing factors include emotional stress, alcoholism, tobacco, fatigue, malnutrition, and general debilitation.

2. *Causative factors.* These are microorganisms which have not been specifically identified. The acute disease develops from a host-parasite imbalance as a result of an overwhelming increase in the number of bacteria and/or lowered patient resistance. Organisms

that have been associated with NUG are *Bacillus fusiformis, Borrelia vincentii,* alpha-hemolytic streptococci, *Bacteroides melaninogenicus,* and other unidentified vibrios, spirochetes, and streptococci. It is interesting that the number of spirochetes is proportional to the amount of inflammation and the amount of necrosis present. Some investigators have observed organisms penetrating the tissue in the lesions of NUG. Others believe that organisms do not invade, but are found on the surface of the wound and are only observed within the tissue because they were incorporated during tissue preparation. Even though a specific organism has not been conclusively demonstrated to produce NUG, it has been well established that microorganisms are the causative agents of the disease. The dramatic response to antibiotics, both topical and systemic, is valid evidence of the disease's bacterial etiology. Once antibiotic administration is stopped, the disease will usually recur unless the predisposing factors have been eliminated.

DIAGNOSIS

Necrotizing ulcerative gingivitis can be diagnosed on the basis of clinical findings alone. The onset of the disease is manifested quite suddenly, and patients complain of severe pain about the teeth or gums. Usually, they cannot determine any one particular area that hurts but say, "My entire mouth hurts," or "All of my teeth hurt." The pain is more intense at the sites of ulceration. The second most prominent symptom experienced by the patient is bleeding gums. Bleeding is often spontaneous, and patients may observe blood on their pillows or notice the taste of blood when they awaken. Patients may also experience marked pain and bleeding while brushing their teeth or when eating. Alcoholic beverages, hot or cold liquids, or spicy foods may be intolerable.

The most characteristic and pathognomonic finding of NUG is ulceration and cratering of the interdental papillae (fig. 21-1). Frequently, the papillae are reduced to punched-out masses of necrotic tissue covered by a gray-white pseudomembrane. Acute pain and bleeding result from the slightest pressure on the area. Ulcerated areas spread by contiguity and by contact. The mucosa of the lips, the jaws, and the palate may be affected, and ulcerated areas may be found on the tongue.

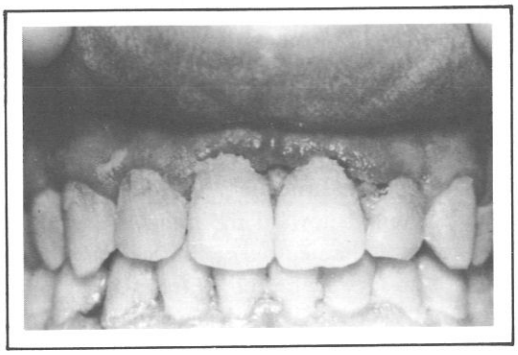

Figure 21-1. Necrotizing ulcerative gingivitis present for 3 days in 21-year-old male whose chief complaint was pain. Note cratering of interdental papillae.

The fetid odor of necrosis is usually present and can easily be remembered by the dental officer, once he has experienced it. However, this distinctive odor is not pathognomonic of NUG but is present in any site of tissue necrosis.

There may or may not be a number of systemic findings. In very acute cases the body temperature may be elevated. There may be headache, general malaise, loss of appetite, and regional lymphadenopathy. The constitutional symptoms seem to parallel the severity of the disease and are usually more pronounced in younger individuals.

DIFFERENTIAL DIAGNOSIS

A number of diseases produce lesions similar to those of NUG. Lesions most commonly mistaken for NUG include those of acute gingivitis, primary herpetic gingivostomatitis, recurrent aphthous stomatitis, desquamative gingivitis, infectious mononucleosis, acute leukemia, agranulocytosis, and the secondary stage of syphilis. It should be emphasized that NUG can occur in conjunction with any number of systemic debilitating diseases. For example, NUG has been observed in cases of infectious mononucleosis, acute leukemia, and uncontrolled diabetes. For this reason, if there has not been a dramatic response to the local treatment of NUG by 72 hours, the patient should be referred for medical consultation.

1. *Acute gingivitis* (fig. 21-2). An intense generalized or even localized acute gingivitis can mimic many of the signs and symptoms of NUG. In gingivitis, pain is not as severe nor as

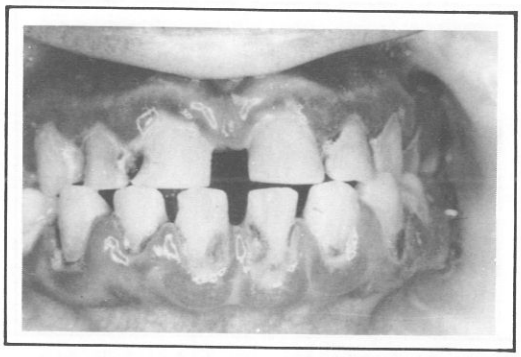

Figure 21-2. Extensive ***generalized gingivitis*** *due to plaque infection in 38-year-old male. Duration was unknown and there was no chief complaint. No signs of interdental cratering could be seen.*

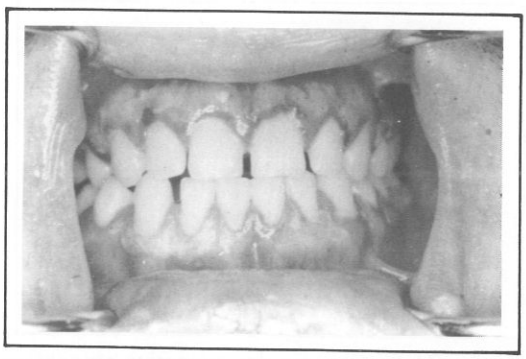

Figure 21-3. Primary acute herpetic gingivostomatitis of 3 days' duration in 18-year-old male, who had no history of previous oral lesions. He was experiencing elevated temperature (102°F), general malaise, regional lymphadenopathy, and extreme intraoral pain. Ulcers were observed on lips, buccal mucosa, and gingiva. There were no signs of interproximal cratering.

persistent and rarely is there spontaneous bleeding. In many patients with acute gingivitis, the interproximal areas and gingival margins are filled with food, plaque, and materia alba. Once this debris is removed and the interproximal areas can be examined, the lack of necrosis and crater formation will verify the diagnosis of nonulcerative gingivitis.

2. *Primary acute herpetic gìngivostomatitis* (fig. 21-3). This disease is characterized by small ulcers with elevated, halo-like margins. The lesions are yellowish and cheesy in appearance and bleed less readily on pressure than does NUG. The disease is accompanied by generalized soreness, which interferes with eating or drinking. The typical interdental crater of NUG is lacking. Patients usually display rather severe constitutional symptoms, with typical herpetic lesions extraorally and intraorally. Diagnosis is based on clinical findings and patient history. Acute herpetic gingivostomatitis usually runs a course of 7 to 10 days. Treatment consists of palliative measures. The patient is placed on warm water rinses, soft diet, and forced fluids. Plaque and superficial calculus are removed to reduce gingival inflammation, which complicates the herpetic gingivitis. If the patient experiences pain on eating, an 0.5% solution of dyclonine hydrochloride is prescribed before meals. It is swished in the mouth for about 2 minutes and then expectorated. This will produce local anesthesia up to an hour. Dyclonine hydrochloride can be used several times daily without fear of toxicity.

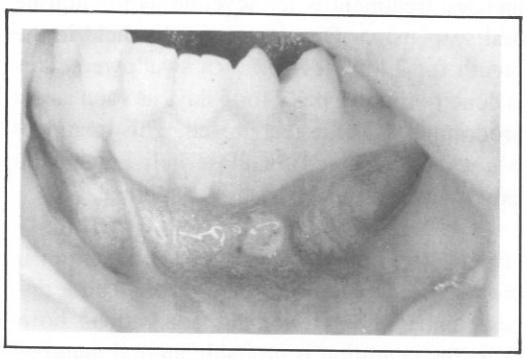

Figure 21-4. Recurrent aphthous stomatitis.

3. *Recurrent aphthous stomatitis (canker sores)* (fig. 21-4). This condition is characterized by single or multiple epithelial erosions, which can occur on the buccal mucosa, lateral margin of the tongue, floor of the mouth, soft palate, marginal gingiva, and pharynx. The ulcers are covered by a gray-white membrane with an erythematous margin and minimal adjacent erythema. The condition is extremely painful, and one or more oral lesions may always be present. Studies indicate that the causative agent may be an L-form of *Streptococcus sanguis*. Common precipitating factors include mucosal and gingival trauma, psychic stress, and endocrine imbalance. In patients who suffer

Figure 21-5. Chronic desquamative gingivitis. This 42-year-old female patient had history of bullous formation, severe burning sensation, and painful gingiva, of 3 to 4 months' duration. This was second episode in past 2 years. There were no signs of interdental craters. Patient could not brush her teeth because of extreme pain.

Figure 21-6. This 42-year-old female had positive diagnosis of infectious mononucleosis. Persistent gingivitis related to plaque formation existed but there was no interdental cratering.

continuously with this condition, the recommended treatment is tetracycline hydrochloride oral suspension. One teaspoonful is held in the mouth for 2 minutes and then swallowed. This is done four times daily for 5 days as soon as the prodromal signs are recognized. This treatment is *not* recommended for those individuals who experience only the occasional aphthae.

4. *Chronic desquamative gingivitis (gingivosis)* (fig. 21-5). This gingival condition is probably a clinical syndrome rather than a disease entity. The etiology is not known; however, the condition is probably an oral manifestation of a bullous dermatologic disease, such as benign mucous membrane pemphigoid or lichen planus. Desquamative gingivitis is most commonly observed in females (17 to 40 years old) and can occur in mild, moderate, and severe forms. In the mildest form there is diffuse, painless erythema of the gingiva. In the moderate to severe form there are scattered red and gray areas involving the marginal and attached gingiva. The gingiva can usually be rubbed off with finger massage (Nikolsky's sign), leaving a bleeding surface. The papillae do not undergo necrosis; therefore, there is no interdental cratering. The patients complain of a burning sensation, thermal sensitivity, and pain on brushing the teeth. The mild form of this condition may be painless, but the severe form is extremely painful. Diagnosis is based on clinical findings and biopsy. Treatment consists of gentle prophylaxis, plaque control, and elimination of all forms of local irritants. In the more severe cases, systemic corticosteroids are used to supplement the local therapy. Topical hormones are often effective supplements to local therapy: For female patients, a cream containing 1.25 mg/Gm of conjugated estrogen; and for males, methyltestosterone ointment, 2 mg/Gm. Some therapists have successfully eliminated the condition by gingivectomy.

5. *Infectious mononucleosis* (fig. 21-6). This benign infectious disease is usually seen in children and young adults. The symptoms include a sudden onset of fever, nausea, headache, vomiting, and swelling and tenderness of the lymph nodes. The patient often complains first of a sore mouth and throat. Orally, there may be diffuse erythema of the mucosa and petechiae. The marginal gingiva and interdental papillae are swollen, inflamed, and bleed upon gentle pressure or spontaneously. There is no ulceration or interdental crater formation, but secondary development of NUG affords a diagnostic challenge. Diagnosis is based on hematologic findings.

6. *Leukemia* (fig. 21-7). Oral manifestations occur with great frequency in patients with leukemia, particularly acute and subacute monocytic leukemia. Clinical changes may vary from a diffuse cyanotic discoloration of the entire gingival mucosa to a tumorous gingival enlargement. The enlargement may be localized or generalized, diffuse or marginal; but in all cases it is associated with local irritants, such as

Figure 21-7. Acute monocytic leukemia in 28-year-old male. Enlargement of gingiva had become progressively worse over past month. Patient complained of mildly painful gingiva that bled easily on contact. He experienced difficulty eating and incising.

Figure 21-8. Agranulocytosis caused by high dosage of aspirin for arthritis. This 42-year-old male had generalized hyperplastic gingivitis but no signs of interdental cratering.

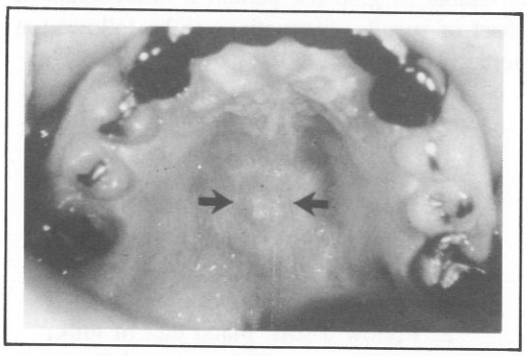

Figure 21-9. Secondary syphilis. This 38-year-old male exhibited midpalatal mucous patch and regional lymphadenopathy. Gingiva was not involved.

plaque, calculus, faulty restorations, and trauma. It is not uncommon to observe the clinical signs of necrotizing ulcerative gingivitis superimposed upon leukemic gingival enlargement. When there is lack of response to local treatment of NUG, a complete blood count, urinalysis, and bone marrow studies are essential to rule out the presence of leukemia and other blood dyscrasias.

7. *Agranulocytosis (malignant neutropenia)* (fig. 21-8). This condition is manifested orally as ulceration and necrosis of the gingiva, which resembles NUG. The ulcers are covered by a gray or gray-black membrane, but there is less inflammation associated with the lesions of agranulocytosis than NUG. Lesions are also observed in the oral mucosa, tonsils, and pharynx. The most common cause is the ingestion of a wide variety of drugs (aspirin, barbiturates, etc.), which may account for the apparent increase of this disease. A differential diagnosis is based on blood studies.

8. *Secondary syphilis (mucous patch)* (fig. 21-9). The oral lesions of syphilis are usually on the tongue, the gingiva or the buccal mucosa. They are usually ovoid or irregularly shaped and surrounded by an erythematous zone. The mucous patch rarely affects the marginal gingiva, and the overlying grayish-white plaque is not detachable. The lesions are usually painless but *highly* infectious. Diagnosis is made by a positive serology and darkfield examination of an affected lymph node.

TREATMENT OF NECROTIZING ULCERATIVE GINGIVITIS

The dental officer should attempt to (1) control the acute bacterial phase, (2) educate the patient in plaque control, and (3) eliminate the predisposing features, both local and systemic. Dentists have used a wide assortment of drugs to treat the acute bacterial phase, but early and vigorous local treatment will produce faster and more dramatic results than any drug therapy. Drugs should never be considered a substitute for scaling and debridement. Antibiotics should be used only when systemic complications are evidenced.

Basic steps in treatment are as follows:

First visit.

1. Remove as *much* calculus, plaque, and debris as possible, as *soon* as possible, and as

gently as possible. Ultrasonic instrumentation is the method of choice because it provides both irrigation and debridement.

2. Instruct the patient in plaque control. Begin patient education and motivation. Write the patient a prescription for a soft brush to use during the acute phase. If he/she has a nylon bristle brush, you may advise him/her to hold the brush under warm water to soften the bristles.

3. Antibiotics may be administered systemically if there is evidence of elevated temperature, lymphadenopathy, and general malaise. Most cases do not require antibiotics. Mild analgesics may be prescribed for pain.

4. Instruct the patient in home care. It is advisable to give the patient a mimeographed sheet of specific instructions to be followed at home. The following list may be used:

Home Care Instructions for the Patient

1. Rinse the mouth vigorously with warm saline solution (1 level teaspoon of table salt dissolved in 1 glass (8 oz.) of warm water) every 2 hours.
2. Follow a soft, bland diet of milk, eggnog, broth, etc. Dietary supplements (Nutrament, Metrecal, Carnation Instant Breakfasts, etc.) are especially useful during this period.
3. Drink plenty of water.
4. Avoid foods that are hard, fried, coarse, spicy, or starchy.
5. Reduce smoking and drinking of alcoholic beverages.
6. Rest as much as possible.
7. After eating, rinse the mouth with warm saline solution.
8. Brush the teeth in the manner prescribed. A soft brush is advisable at this stage.
9. Return in 24 hours.

Second visit.

1. Check oral hygiene and review plaque control procedures.
2. Continue removal of calculus, plaque, and debris.
3. Provide prophylaxis.
4. Have patient return in 24 hours.

Third visit.

1. Check plaque control. Review procedures, if indicated.
2. Continue elimination of all irritants. This includes all calculus, overhanging margins, open contacts, etc.
3. *If* the tissues have not responded dramatically by the third visit (48 to 72 hours), evaluate for systemic factors (leukemia, infectious mononucleosis, etc.). Refer for medical consultation, if necessary.
4. If improvement is apparent, make an appointment for reevaluation in 1 week.

Fourth visit.

1. Check plaque control.
2. Check for calculus.
3. Evaluate for further periodontal therapy.

NUG responds rapidly and dramatically to local therapy and effective plaque control. As a result, some patients and clinicians become somewhat complacent about the severity of the disease. It is important to remember, and to caution the patient accordingly, that unless the treatment is continued to completion, NUG is likely to recur. Likewise, uncontrolled NUG can result in localized osteonecrosis, extensive soft tissue destruction, or even Ludwig's angina.

Hypersensitivity

Hypersensitivity is often a management problem for both patient and dentist. This is true despite the wide variety of medicaments and desensitizing paraphernalia available.

ETIOLOGY

Hypersensitivity of exposed dentin can occur when dentinal tubules are exposed either by caries, fracture, periodontal disease, or periodontal instrumentation. Traumatogenic occlusion is also a frequent cause of hypersensitivity. Under such circumstances, thermal stimuli (hot or cold foods), chemical stimuli (sweet or sour foods), and tactile stimuli (toothbrushes and dental instruments) can excite a painful response. It is most discouraging to the patient—and futile for the dentist—to insist upon vigorous plaque control when the procedures are painful.

TREATMENT

Hypersensitivity can be controlled by eliminating the etiologic factors and by using desensitizing agents. One particularly effective agent is an ophthalmic suspension which contains prednisolone acetate.

Immediately before the prednisolone suspension is applied, the sensitive teeth are cleaned with flour of *pumice* and dried by a gentle flow of warm air, or by a cotton pellet if they are extremely sensitive. Some clinicians prefer to place rubber dam prior to application of the drug. It is applied continuously for 2 minutes with pledgets of cotton. There should be immediate relief of symptoms. The shelf life of the suspension is approximately 3 months.

The prednisolone suspension is most effective in the treatment of sensitivity due to incisal/occlusal fractures, extensive occlusal adjustment or odontoplasty, periodontal surgery, and scaling and root planing. It is often effective in sensitivity related to gingival recession but may require several applications. In persistent cases, it may be necessary to have the patient use a desensitizing toothpaste after prednisolone therapy. The routine use of a dentrifrice containing sodium monofluorophosphate or strontium chloride has occasionally proved beneficial in the control of persistent cervical hypersensitivity.

Another desensitizing agent which is effective in controlling hypersensitivity is dibasic calcium phosphate plus 30% stannous fluoride. The teeth are polished with pumice and the mixture is burnished into the sensitive areas with a porte polisher. A cavity varnish such as Copalite can be placed over the areas to prevent rapid loss of the desensitizing agent.

Primary Periodontal Traumatism

DEFINITION

Primary periodontal traumatism is the condition in which tissue injury (trauma from occlusion) is produced in an otherwise normal periodontium by excessive occlusal force (traumatogenic occlusion). Secondary periodontal traumatism is the condition in which relatively normal occlusal forces produce injury in a weakened periodontium. Microscopically, both conditions may appear the same. The periodontal ligament demonstrates necrosis, hemorrhage, thrombosis of blood vessels, and dissolution of principal fibers; and there is bone loss and widening of the periodontal ligament space. It should be reemphasized that excessive occlusal stress does not initiate gingivitis and pocket formation.

SIGNS AND SYMPTOMS

Traumatogenic occlusion may result in pain on chewing, tenderness to percussion, and thermal sensitivity. The traumatized tooth is often mobile. In such instances, the pain is localized and is usually associated with the recent insertion of a restoration or a dental appliance, with parafunctional habits, or with a recent traumatic injury to the jaw or teeth. If the tooth is vital, it is frequently hypersensitive to electrical stimuli.

If excessive occlusal forces are of long duration, the tooth or teeth are mobile, but usually there is little or no pain when chewing and little response to percussion. Migration of the traumatized tooth is frequently evidenced. The patient complains of increasing thermal sensitivity and occasionally a dull aching sensation. Diagnosis can often be made by placing the index finger over the mobile tooth and having the patient glide into working and nonworking excursions. The tooth will move in and out of alinement as the excursions are accomplished (fremitus).

TREATMENT

Treatment of primary periodontal traumatism consists of eliminating the etiologic factor or factors. This may entail selective grinding or removal of the irritating appliance. Where the cause was a severe blow, it may be necessary to splint the teeth until the acute symptoms have subsided. Bite guards have been used very effectively to manage this problem. Once the traumatogenic force is eliminated, the potential for complete reversibility of the lesion is good. In most cases, root sensitivity will disappear after adjustment of the occlusion. Desensitizing solution is often applied to afford immediate relief of the sensitivity and to encourage plaque control.

Temporomandibular Joint Pain Dysfunction Syndrome

ETIOLOGY

Injury to the temporomandibular joint and the muscles related to the function of the joint may result from an extrinsic source such as a traumatic blow, or from an intrinsic source such as a muscle spasm. It is not within the scope of this chapter to discuss the multitude of signs, symptoms, etiologic factors, and therapies proposed for TMJ dysfunctions; however, it is important that all dentists have a basic understanding of this extremely painful and demoralizing condition. It is generally, but not universally, agreed that the cause of the dysfunctional pain is a combination of psychic tension and occlusal disharmony. These etiologic factors acting together result in hyperactivity and pain in the masticatory muscles and dysfunction of the jaw. There is no evidence to show organic changes of the joint, except for degenerative arthritis; consequently, the sequelae are similar to those that occur in any joint complex after a long-standing functional disorder.

SYMPTOMS

The clinical syndrome is associated with five symptoms, which may vary from patient to patient:

1. Pain in the region of the TMJ, ear, face, neck.
2. Cracking or popping noises associated with TMJ movement.
3. Limitation or deviation of mandibular movement (muscle spasm).
4. Subluxation or dislocation of the mandible.
5. Difficulty in mastication of food.

TREATMENT

Many forms of therapy have been proposed for this problem and some success has been reported with most of them. Studies continue to indicate that psychological factors play a major role in the onset of most TMJ problems. Consequently, many clinicians are adopting techniques which do not result in irreversible alterations of teeth, muscles, or joint structures, yet afford successful treatment. The following approach is recommended as one concerted but effective method of management of TMJ pain dysfunction syndrome:

1. Talk and listen to the patient. Diagnose the problem. Always take a positive approach to patient management. Assure the patient that the problem can be corrected.
2. Make an impression for a bite guard as soon as possible.
3. When there is extensive trismus, prescribe a tranquilizer until the trismus subsides. Diazepam (5 mg tablets), is very specific for this problem and is the drug of choice, if one is needed. It is administered by asking the patient to take one-half tablet in the morning and one-half tablet in the evening.
4. Instruct the patient to:
   a. Limit mandibular movements
   b. Eat a soft diet; avoid incising foods; chew bilaterally.
   c. Avoid yawning. This can be accomplished by clasping hands on the back of the neck and pulling forward with the arms when the urge to yaw arises. This forces inspiration and will usually prevent yawning.
   d. Apply warm, moist heat as often as possible (4 to 6 times daily).
   e. Avoid teeth clenching, pipe smoking, fingernail biting, etc.
   f. Relax by walking, jogging, playing tennis; taking hot showers, hot baths, and sauna baths; etc.
   g. Sleep on his back.
5. Adjust and insert the bite guard as quickly as possible.
6. Tell the patient to wear the bite guard 24 hours daily, removing it only to accomplish plaque control.
7. Once the acute symptoms have subsided, ask the patient to continue to wear the bite guard during sleep and during periods of stress.

22

Periodontal considerations in restorative dentistry

Tooth preparation
Narrow band of attached gingiva
Wedges
Retraction strings
Contours
Marginal ridge
Embrasures
Fixed partial dentures
Removable partial dentures

In this chapter, the term "restorative dentistry" encompasses the restoration of individual teeth to normal contour and function, and the restoration of the dental arches to integrity and improved function. All phases of restorative dentistry are dependent on the establishment and maintenance of periodontal health to ensure lasting benefit. Considerations relating to operative dentistry and fixed and removable prosthodontics are included here.

TOOTH PREPARATION

Permanent damage to the periodontium should be avoided during operative procedures, and the completed restoration should not interfere with plaque control, should not promote food impaction, and should be compatible with the patient's optimal occlusion. It is axiomatic that procedures should be as atraumatic as possible. Restorations should be cleansable by the patient, a feature which requires marginal excellence and a smooth finish.

NARROW BAND OF ATTACHED GINGIVA

The quality of the gingiva should be considered when the margin of a crown or Class V restoration is to be placed subgingivally. If the zone of attached gingiva is inadequate or minimal, the gingival margin is likely to recede after margin preparation, gingival retraction, impression making, and placement of temporary restorations. The problem can be avoided by placing margins well away from the gingiva (fig. 22-1),

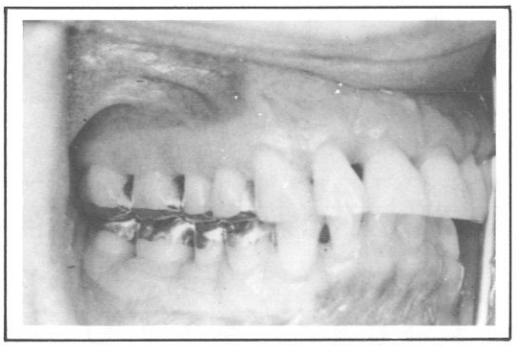

Figure 22-1

but the requirements for retention and the location of caries frequently preclude this approach. Another solution would be to establish a somewhat wide band of attached gingiva at the site before restoration. As mentioned in chapter 16, ''Management of soft tissue: flaps and free grafts,'' this gain in attached gingiva can be established with free grafts or laterally positioned flaps. If a convenient donor site is available and if the only problem is to establish attached gingiva at the operative site, the free graft is the best choice.

WEDGES

When a matrix is placed, caution is needed to prevent laceration of tissue. Carelessly placed wedges may cause unintentional papillectomy. Use of a rubber dam helps prevent such damage, but the rubber dam clamps can be destructive if improperly positioned on tissue rather than teeth.

RETRACTION STRINGS

Impregnated strings used for gingival retraction can injure the gingiva if left in place too long or if the gingival tissues are inflamed prior to retraction. The effects of the chemicals in the retraction strings cannot be controlled, and pressure retraction of the gingiva with chemical-free strings is recommended whenever possible. The vasoconstrictor (8 percent racemic epinephrine) in an impregnated retraction string is rapidly absorbed and can cause a transient elevation of blood pressure and blood sugar. Vasoconstrictor impregnated strings are contraindicated in patients with coronary disease, diabetes, or hyperthyroidism.

CONTOURS

Tooth contours should be such that food is shed away from the gingiva during mastication. These ''shedding'' contours are in the *occlusal* thirds of the crowns of posterior teeth and the *incisal* thirds of the facial and lingual surfaces of the anterior teeth. Although the concept on tooth contours that they should ''permit proper stimulation of the gingiva by fibrous food'' is an old and popular one, teeth are naturally shaped to *prevent* food from contacting the gingiva during the masticatory occlusal stroke. Overcontouring, however, may encourage stagnation of food in the gingival sulcus and will interfere with plaque control procedures and the mechanical cleansing action of the cheek against the tooth surface. Restoration of *normal* tooth contour should be a major objective of the restorative dentist (fig. 22-2).

MARGINAL RIDGE

Approximating marginal ridges should be even in height, and well-defined occlusal fossae should be developed in the restoration. Such refinements help prevent the wedging of food interproximally. Inconsistent relations of the marginal ridges is the factor most frequently associated with food impaction and corresponding infrabony pocket formation. When the maxillary anteriors are restored with full coverage, lingual cingula should be developed to shunt incised food away from the palatal gingival margin.

EMBRASURES

In health, the gingival embrasure is occupied by the interdental papillae. In periodontal disease, bone and soft tissue are lost, and the interdental space is opened. Preferably, restorations will be placed that preserve the morphology of the crown and root and retain the enlarged embrasure. The height, width, and faciolingual depth of the embrasure must be maintained. The proximal surfaces of restorations should:

1. Taper away from the contact area. Broad contact areas crowd out the interdental papillae and reduce access for plaque control. The papillae become prominent and entrap food debris, and this action leads to inflammation and pocket formation.

2. Be slightly convex. Concave contours make plaque control difficult.

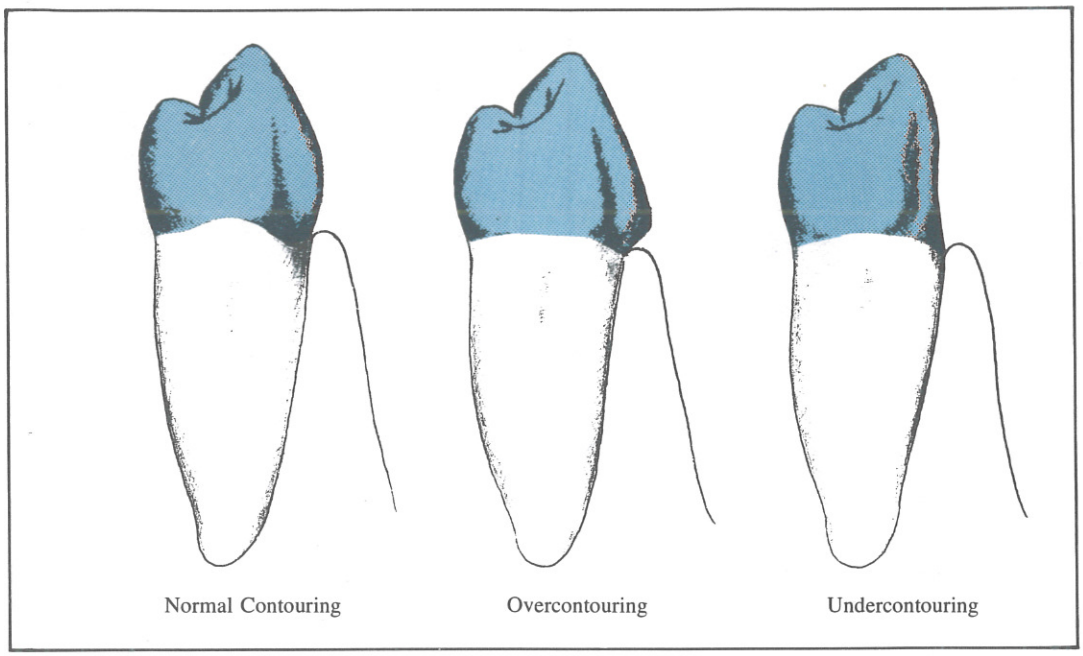

Figure 22-2

3. Contact faciolingually to a degree that prevents food impaction.
4. Be as smooth as possible.

FIXED PARTIAL DENTURES

When missing teeth are replaced by fixed partial dentures, the abutment teeth must carry an increased burden, which can further weaken abutments already weakened by periodontal disease. This burden may be eased somewhat by (1) decreasing the buccolingual width of the pontics, (2) directing occlusal forces primarily along the long axes of the teeth, and (3) including a sufficient number of sound teeth in the prosthesis to distribute occlusal forces.

Fixed partial dentures complicate plaque control by the patient. Where esthetics permits, the use of sanitary pontics (fig. 22-3) or bullet-shaped spheroidal pontics (fig. 22-4) provides better access for plaque control. In the anterior region, the modified ridge lap pontic (fig. 22-5) meets esthetic demands and yet can be readily cleansed. The patient will require auxiliary devices, such as bridge cleaners or twisted wire "needles," to facilitate threading floss or yarn though the embrasures.

Fixed partial dentures should be so designed that every surface can be readily cleansed. This implies convex surfaces and open embrasures.

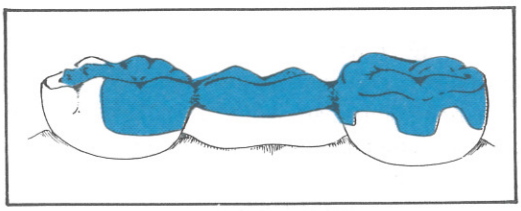

Figure 22-3

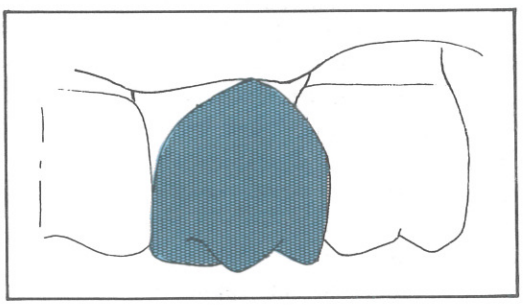

Figure 22-4

The position of solder joints can be critical; they should be located as far away from the gingiva as possible, to permit proper embrasure form.

Overhanging margins lead to damage of the periodontium, primarily because they provide breeding places for microorganisms which are inaccessible to the patient's plaque control devices.

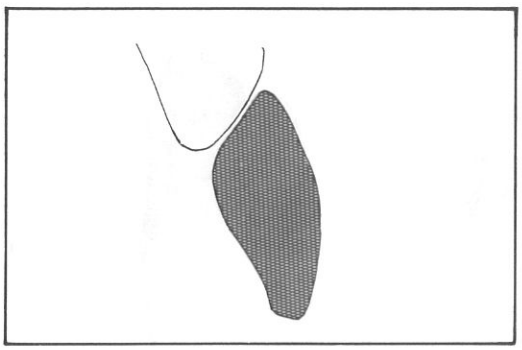

Figure 22-5

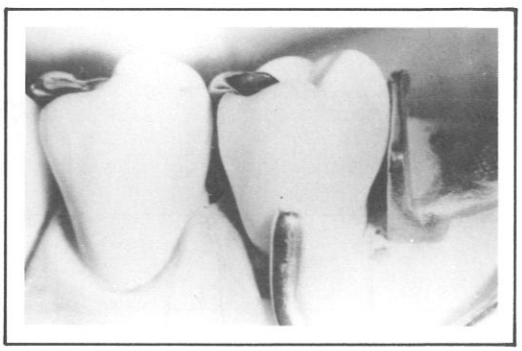

Figure 22-6

Care must be taken to remove all excess cement from the gingival sulcus and/or pontic. Retained cement particles act in the same manner as calculus and can result in plaque retention, inflammation, and pocket formation.

REMOVABLE PARTIAL DENTURES

The design of removable partial dentures should contribute to masticatory efficiency and esthetics without damaging the remaining teeth and supporting structures. The need for excellence in design is particularly acute with distal extension removable partial dentures. In these appliances, abutment teeth are expected to receive and survive heavy, multidirectional forces. Such concepts as the altered-cast technique, the mesioclusal rest, the I-bar clasp, the guiding plane, and the meticulous adjustment of the framework are all based on consideration for the remaining teeth (fig. 22-6).

Regardless of the restorations placed, all patients should be instructed and motivated to control bacterial plaque. Those who received fixed or removable partial dentures should be given specific instructions concerning plaque control problems they will encounter. Patients must be instructed to routinely remove plaque from all surfaces of a removable appliance as diligently as they cleanse their teeth. Frequent evaluation is necessary to ensure patient effectiveness and cooperation.

23

Problems and failures in periodontal therapy

General causes of failure
- Failure to instruct patient adequately in plaque control
- Failure to formulate and operate from a simple, comprehensive written treatment plan
- Failure to survey and correct common local factors
- Failures related to diagnostic deficiencies
- Failures in approach to periodontal surgery
- Failures in surgical technique
- Failure to maintain an effective patient recall system

Summary

It is appropriate in this syllabus to consider the prospect of failure in the management of periodontal disease. Unhappily, many dentists anticipate therapeutic failure even as they initiate treatment. Indeed, because of the complex etiologies and treatment of periodontal disease, many clinicians consider failure as a very likely possibility. Too frequently, failure is considered inevitable and the disease mysterious, leading inexorably to the edentulous state. Moreover, few dentists, other than periodontists, ever examine the causes of failure. Most dentists remain content, somehow, to accept "systemic inadequacies" and "patient oral hygiene inadequacy" as insurmountable impediments to successful periodontal therapy.

Most periodontal therapeutic failures, however, are far from mysterious. Since most periodontitis is caused by local factors, unsatisfactory therapy generally can be attributed directly to failure to identify and eliminate those local factors. In everyday practice, relatively few cases have their origins in bizarre systemic derangements.

Of course, failure may be total or partial, reversible or irreversible. Areas that do not respond satisfactorily to initial treatment methods, for instance, probably should not be classified as failures. Frequently, even surgical corrections must be planned as multistage procedures to achieve both pocket elimination and optimal tissue architecture.

Effective periodontal therapy implies comprehensive patient management. Unlike carious lesions, which usually require placement of a single restoration, periodontal problems rarely respond to one-step treatment. What appears to be success on evaluation after a few weeks, may prove to be a total failure on scrutiny after a year.

This poses a special problem in naval practice. Naval dental officers are seldom able to evaluate cases over a long period of time. Consequently, the failure rate may be higher than anyone realizes.

This, then, is why a consideration of "failure" is so vital.

General Causes of Failure

The following are the most common "mysterious" causes of failure in periodontal treatment. Most of these have been discussed in other chapters, but are emphasized here to underscore the importance of their relationship to failure.

FAILURE TO INSTRUCT PATIENT ADEQUATELY IN PLAQUE CONTROL

Failure to instruct a patient adequately in plaque control is considered by most periodontists to be the principal reason for failure. This problem may have several roots:

1. The practitioner may be overconfident of his knowledge of advanced plaque control philosophy, techniques, and effectiveness.
2. The practitioner may fail to communicate with, and/or to motivate, the patient effectively; fail to reinforce previous instructions; or fail to evaluate the adequacy of previous instructions.
3. He may fail to utilize disclosing media for plaque visualization.
4. He may fail to give adequate demonstrations of the use of the toothbrush, floss, yarn, and other plaque control devices.
5. The crux of the problem may be his failure to believe—and thus his failure to communicate—that the patient's personal plaque control efforts are just as vital to periodontal therapy as the practitioner's procedures. A dentist who does not himself practice rigid daily plaque control will not succeed in motivating patients to become effective "cotherapists" in treating their own disease.

The *patient* may lack the motivation, the dexterity, or the mentality to accomplish effective plaque control. Under these conditions, periodontal therapy will quickly fail.

FAILURE TO FORMULATE AND OPERATE FROM A SIMPLE, COMPREHENSIVE WRITTEN TREATMENT PLAN

Failure to allow sufficient time in the plan to evaluate the adequacy of current therapy unquestionably accounts for many treatment failures.

FAILURE TO SURVEY AND CORRECT COMMON LOCAL FACTORS

The failure to survey and correct common local factors is universally regarded as a prime cause of unsatisfactory therapy. The principal reason why the following local factors are significant is that, because of them, plaque tends to accumulate, its easy removal is prevented, or food impaction is induced or permitted.

1. Calculus.
2. Open contacts (verify visually and with floss).
3. Rough restorations and overhanging margins.
4. Uneven, sharp, or nonexistent marginal ridges.
5. Plunger cusps (can induce wedging of opposing teeth and produce food impaction).
6. Overcontoured crowns, uncleansable pontics of absurd, antibiologic design, and removable partial dentures that are damaging to abutment teeth. In general, it can be stated that, sooner or later, anything less than absolutely fastidious execution of restorative procedures can be demonstrated to be a compromise, periodontally.

FAILURES RELATED TO DIAGNOSTIC DEFICIENCIES

1. Failure to obtain an adequate health history and failure to discern possible systemic influences on periodontitis or on patient management can have disappointing or even disastrous consequences.

2. Dentists frequently fail to use a calibrated probe to evaluate the depth and location of pockets. On the contrary, ironically, they may be meticulous in their use of an explorer to search for caries.

3. Similarly, failure to use a special furcation probe in furcation areas may leave areas of serious involvement undetected.

4. Failure to obtain adequate radiographs or to adequately interpret radiographs are unpardonable omissions for the conscientious therapist.

5. Failure to note such anatomic characteristics as abnormal frenum influence, width of attached gingiva, or root prominences with associated dehiscence and fenestration deformities may have unfortunate consequences postsurgically.

6. Failure to identify and correct *traumatogenic* occlusal relationships, including centric pathway prematurities, nonworking side contact, parafunctional habits, plunger cusps, etc., will compromise therapy. Routine examinations of occlusion on every patient must be emphasized.

7. Many total periodontal failures can be laid to ill-considered or haphazard periodontal appraisal; namely, failure to recognize and remove hopelessly involved teeth.

8. Failure to consider the influences of periapical or pulpal disease on periodontal therapy can result in unexpected complications or unexpected loss of strategic teeth. The intimate pulpal-periodontal relationship has been increasingly appreciated in recent years.

FAILURES IN APPROACH TO PERIODONTAL SURGERY

The benefits of periodontal surgery are well documented. Properly planned and executed, these procedures can facilitate plaque control by eliminating pockets and improving contours and other anatomic relationships. Casually conceived, however, surgery can result in the most frustrating of failures.

1. Selection of inappropriate surgical techniques at the outset can only lead to unacceptable results. Unfortunately, the term "gingivectomy" has become synonymous with all forms of periodontal surgery.

2. Excision of all attached gingiva, leaving margins of alveolar mucosa, results in an untenable relationship. If such a situation is anticipated, a flap procedure rather than a gingivectomy is indicated. Neither time, "functional stimulation," nor prayer will convert alveolar mucosa into acceptable marginal tissue.

FAILURES IN SURGICAL TECHNIQUE

1. Inadequate debridement of deposits, granulomatous tissue, amalgam particles, etc., can cause delayed healing or surgical failure.

2. Ragged, torn tissue on flap edges resulting from careless design, dull or improper instruments, and generally sloppy technique can result in delayed healing and undesirable contours.

3. Failure to use aseptic technique can result in postoperative infection and surgical failure.

4. Failure to approximate wound edges to ensure healing by primary intention may result in failure of new attachment procedures.

5. Failure to stabilize grafts and flaps leads to displacement, undesirable contours, or involution, and to unnecessary sequestration of bone—and pain!

6. Failure to use pressure coaptation of flaps and grafts after suturing may result in formation of a large fibrin clot, resulting in downgrowth of the epithelial attachment and bulbous contours.

7. Inadvertent forcing of periodontal dressing underneath flaps results in permanent tissue defects.

FAILURE TO MAINTAIN AN EFFECTIVE PATIENT RECALL SYSTEM

One of the major reasons for failure of periodontal therapy is the lack of an effective recall system. Periodontal patients must be reevaluated every 3 to 4 months. This permits a careful examination of the tissues and an opportunity to determine the effectiveness of the patient's plaque control. If there is indication of tissue breakdown and/or laxity in plaque control, reinforcement of basic principles will usually reverse the disease process. Insufficient numbers of facilities, periodontists, and trained auxiliaries, and also the transiency of patients and doctors, complicate the establishment of an effective recall system in the Navy. Recurrence of periodontal disease, then, becomes a reality, unless all dental officers are willing to assume the responsibility for the recall, reevaluation, and reinforcement of the periodontal patient.

Summary

An analysis of failure in periodontal therapy reveals a many-factored problem. Most failures, whether partial or complete, can be attributed to inadequacies of plaque control, diagnosis, surgical techniques, or maintenance. The greatest single cause for periodontal disaster, however, results from failure of the therapist to educate and motivate the patient to practice effective plaque control.

The periodontal therapist must also assume responsibility for *total case management*. He is the quarterback. In addition to periodontal treatment, he must coordinate prosthetic support and medical requirements and establish a rigid recall system.

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